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EXAMINATION IN THE HISTORY OF OTOLOGY.

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1. Demonstrate how the folklore of the ear supports the thesis of the similarity and continuity of primitive medicine in all parts of the world. Cite ancient incantations, old peasant songs, and current superstitions for the cure of earache.

2. Prepare a thesis on: Fashion as illustrated in the earrings of savage and superior races.

3. Write an essay on The Ear in Art, with illustrations.

4. Cite Babylonian birth-prognostications of aural interest, such as: "When a woman gives birth to a child with a lion's ear, there will be a strong king in the land." An Assyrian tablet-fragment describes acute inflammation of the middle ear as "fire in the heart of the ear." Among the thousands of baked clay tablet-fragments, cite those dealing with the ear. Use these collected quotations as the basis of an essay on: Otology in Mesopotamia.

5. What evidence of aural pathology has been found in mummies and in the skulls of predynastic Egyptians? What proof have we that the ancient Egyptians suffered from mastoiditis? Discuss the otologic cases in the Edwin Smith Papyrus (17th century B.C.) and the prescriptions for disorders of the ear in the Papyrus Ebers (16th century B.C.).

6. Prepare a thesis, with bibliography and illustrations, on: History of the anatomy of the ear, from Alcmaeon of

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Crotona (6th century B.C.) to Gustav Alexander of Vienna (20th century A.D.).

7. Write on: The otological knowledge of Hippocrates (460-370 B.C.). Why were fractures of the ears frequent among the ancient athletae? Why did Hippocrates teach that neither bandage nor plaster should be applied to a broken ear? Why did he say that suppuration of the ears is more deeply seated than is usually supposed? How did he regard a discharge of pus from the ears of the young? What books in the Corpus Hippocraticum discuss deafness?

8. Write on: A history of error in otology — mistaken theories, harmful drugs, and useless operations.

9. Write on: The deaf in antiquity, as revealed in Greek and Latin literature.

10. Prepare a list of drugs recommended for disorders of the ear by the father of materia medica, Dioscorides (1st century). Point out which of these drugs are employed in otiatrics at the present time.

11. Write a comparative study of: The otological instruments described in the writings of Celsus (1st century), Galen (2nd century), Paulus Aegineta (7th century), Abul Kasim (10th century), Guy de Chauliac (14th century), Ingrassia (16th century), Schelhammer (17th century), Valsalva (18th century), Toynbee (19th century), Bárány (20th century).

12. From the copious writings of Galen, piece together a series of dramatic episodes on: Galen as an otologist. For example, we enter his iatreion, and see him as he applies euphorbium and nard with the ear-spoon or auriscalpium; we follow him to his operating-room, and observe him incising behind the ear in a case of caries, carefully scraping out the necrosed mass through the incision. As the patient is removed to the sick-room to rest, another comes into the iatreion, perturbed and voluble, but Galen smiles at his worry. From his cabinet, Galen selects a clyster auricularis, and instills blackberry juice into the ear of the Roman senator; he talks, and his voice rises in indignation: "I will give you a prescription,

but you must wait for the morning before you receive the remedy. For the lamps are now lit, and every drug-shop on the Via Sacra has closed its doors. What do the medicamentarii care for a patient's life, even the life of one of the fathers of the city? Nothing interests them except pleasure and money. They are no better than the physicians. Do you know the difference between physicians and robbers? The robbers commit their misdeeds in the mountains, while the physicians commit their misdeeds in the capital of the world. All my life I have suffered from a strange disease: I am fond of work, and am zealous for truth. Ah, those unguent-sellers! They know all about the herbs that come from Crete, but are ignorant of those that grow on the very outskirts of Rome. They like exotic plants that come from afar, and are difficult to obtain, so they can charge fantastic prices. But see, this juice of the blackberry, picked in a Roman garden, has done its work, and here is the worm I have extracted from your ear." We may rest assured Galen saw to it that no patient left his office without the conviction that he had just been treated by the greatest physician since Hippocrates.

13. Assume that you are to edit a volume of about 300 pages entitled: Selected Readings in Otology. What authors and what selections would you include?

14. In Galen's time the auditory nerve was called the fifth cranial nerve. When did it become known as the eighth? When was it recognized that the auditory nerve consists of two separate nerves, the cochlear and vestibular roots? Name the main investigators of the anatomy and physiology of the nervus acusticus, with the date of their publications?

15. Trace the jurisprudence of deaf-mutism from the Code of Justinian to the Commentaries of Blackstone.

16. What is the oldest description of otitis media? mastoiditis? hematoma auris?

17. What medical incunabula are of otologic interest?

18. Write an essay on one of the following subjects: (a) Medieval saints who cured the deaf; (b) The dawn of

the education of the deaf; (c) British teachers of the deaf in the seventeenth century; (d) A comparison of Jacob Rodriques Pereire and Charles Michel de l'Epée.

19. Write an essay on one of the following themes: (a) Origin and development of the concept of otitic meningitis; (b) History of otitis media purulenta acuta; (c) History of otitis media catarrhalis chronica; (d) Valsalva's experiment and its influence on the development of otology; (e) Artificial membrana tympani, past and present.

20. Identify the works of the French Renaissance in which these passages occur: (a) A young Roman gentleman, encountering by chance at the foot of Mount Celion a beautiful Latin lady named Veronica, who from her very cradle upwards had been both deaf and dumb, very civilly asked her . . . (b) . . . what is whispered in the ear is ere long proclaimed from the housetop.

21. Write a sketch of the career of the deaf Renaissance painter whose genius earned him the title of the Spanish Titian, and whose affliction brought him the nickname of El Mudo (The Mute).

22. The two chief members of the Pleiad of the Renaissance (the seven stars of French poetry) were deaf. One of these poets dedicated to the other a Hymn to Deafness: translate these remarkable verses into English prose.

23. Write on: Italian leadership in the anatomical investigation of the ear, from the forerunner Berengario da Carpi (-1550) to Antonio Scarpa (1752-1832).

24. Trace the development of the battle between the manual method and lip-reading.

25. Assume you are a priest in the seventeenth century: a congenital deaf-mute, hitherto excluded from communion because of his handicap, has been taught to speak by the method of Juan Pablo Bonet. Since the deaf-mute is no longer mute, would you permit him to celebrate mass? Write your opinion in the style of the period.

26. Who was the first to declare the importance of teaching the deaf to read and write? Who was the first teacher of the deaf? Who wrote the earliest document on the edu-

cation of the deaf? Who devised the first alphabet for the deaf? Who wrote the earliest text on articulation instruction? Who wrote the first book on lip-reading? Who is called the Apostle of the Deaf?

27. Compile: A list of Shakespearean references to the ear. Add a commentary, for example: Hamlet is remarkable for the frequency of its references to the ear, aside from the fact that the play, and the play within the play, turns upon the pouring of poison into the king's ear. What is Shakespeare's nonce-word, hebenon? How often is the organ of hearing mentioned in the plays? in the poems? How does Shakespeare's otology compare with his general medical and surgical knowledge?

28. In what drama did the famous actor, John Philip Kemble, play the part of Abbé de l'Epée? Compile a list of novels, dramas, and melodramas, in which a deaf-mute is one of the characters.

29. Write a brief sketch of the author of the first treatise on otology (1683). What other works on otology were written in the seventeenth century?

30. Write on: Jonathan Swift (1667-1745) as a victim of Ménière's disease.

31. "Reynolds arrived in London on 16 Oct. 1752, greatly developed as a man and an artist, but with two permanent physical defects, the scar on his lip from the accident at Minorca, and deafness contracted from the cold of the Vatican while copying Raphael" (Cosmo Monkhouse). From letters, diaries, and poems of the period, and from subsequent biographies, collect the references to the ear-trumpet of England's greatest portrait-painter. Use this material for an essay on: The deafness of Sir Joshua Reynolds (1723-1792).

32. What important contributions to otology were first published in the Philosophical Transactions (1741-1801) of London?

33. Who invented the speaking-trumpet? ear-trumpet? tuning-fork? otoscope? otophone? acoumeter?

34. Name the authors of the following: (a) Where more is meant than meets the ear; (b) Deaf, giddy, helpless, left

alone; (c) Thy voice was at sweet tremble in mine ear; (e) Let me hint in your organ auricular. — Cite five other poems which contain references to the ear.

35. What laymen did much to advance otology? For example, what postmaster is famous in otology, and why?

36. Name the educators of the deaf to whom monuments have been erected. Illustrate your paper with photographs of these monuments.

37. Assume you are a British judge in the eighteenth century, and the following case comes before you: A rape has been committed on a girl who is a deaf-mute, but the counsel for the defendant argues that the plaintiff did not cry out when attacked, and moreover her testimony is without value since it cannot be confirmed by an oath. State your opinion of the case in the legal phraseology of the period.

38. What is the acoustic importance of the experiment of the Italian musician, Giuseppe Tartini (1692-1770)? Why did a French philosopher call this experiment "a phenomenon of the nerves, a sort of normal hallucination of the sense of hearing, analogous to optical illusions"?

39. Construct a one-act play on any of the following themes: (a) The Nottinghamshire clergyman, William Holder, teaching the deaf-mute, Alexander Popham, to speak; (b) Samuel Johnson pays a visit to Thomas Braidwood's school for the deaf and dumb in Edinburgh; (c) The opening day of James Yearsley's aural institution in Sackville Street, Piccadilly; (d) William Robert Wills Wilde, founder of otology in Ireland, reading the proofs of his Aural Surgery; (e) The Baroness Mayer de Rothschild establishing the Association for the Oral Instruction of the Deaf and Dumb under the direction of William Van Praagh, the Dutch-born pioneer of lip-reading in England.

40. What French freethinker and what French abbé wrote letters on the deaf? What Irish satirist wrote verses on his own deafness? What English essayist wrote a Chapter on Ears? What American poet wrote a sonnet to a deaf girl?

41. What authors have emphasized the importance of otology in military medicine?

42. What authors have written monographs on the excision of the ossicles? To what extent do recent works on the subject differ from the pioneers?

43. Who pointed out the relationship of sea-sickness to the semicircular canals of the ears? Write on: The history of sea-sickness.

44. Who was the first to realize that otosclerosis is not a sclerotic process? Write on: The changing conceptions of otosclerosis, with a survey of early and recent doctrines.

45. Review the first edition of: Anatomy of the human ear, with a treatise on its diseases, the causes of deafness and their treatment (London, 1806), by John Cunningham Saunders (1773-1810), state the main changes that were made in the posthumous editions, and give an estimate of the position of the author in the history of otology.

46. What deaf English authoress wrote about forty books, hundreds of articles and an autobiography in two volumes?

47. Auguste Comte, in his Cours de philosophie positive (1830-1842), as translated by Harriet Martineau (1853), wrote: "We have not yet any fixed ideas as to the way in which intensity of sound may be estimated; nor even as to the exact meaning of the term. We have no instrument which can fulfil, with regard to the theory of sound, the same office as the pendulum and the barometer with regard to gravity, or the thermometer and electrometer with regard to heat and electricity. We do not even discern any clear principle by which to conceive of a sonometer. While the science is in this state, it is much too soon to hazard any numerical law of the variations in intensity of sound." If Comte were writing today, to what extent would he modify his statements?

48. Write on: A study of the partial deafness of Arthur Schopenhauer (1788-1860).

49. Discuss, with quotations from the original paper: The Czech physiologist Jan Evangelista Purkyne (1787-1869)

— his work on the semicircular canals of the internal ear, and his influence on the concept of equilibrium and vertigo.

50. The work of Blumenbach and Darwin made it known that cats and dogs with white hair and blue eyes are deaf. What is the explanation of this phenomenon? Who reported more recent cases? Write an historico-anatomical essay on: Deafness in animals, citing cases, and concluding with a bibliography.

51. What British otologist is quoted in the opening chapter of Charles Darwin's Descent of Man? What portrait-sculptor modeled and drew for this chapter a human ear showing a point projecting from the helix?

52. Define the Aztec ear, Blainville ear, Cagot ear, Darwin ear, Dionysius ear, Morel ear, Stahl ear, Venus ear, Wildermuth ear.

53. Who was the first to describe auditory speech memories? At what period was it known that the auditory speech center is situated on the left side in right-handed individuals, and on the right side in the left-handed? Who contributed to our knowledge of auditory aphasia? Contrast the opposing views of Paul Broca (1824-1880) and Pierre Marie (1853-1929) on the auditory speech center and the motor speech center.

54. Write a brief sketch of the Italian physician who treated Beethoven for otosclerosis. To what extent, if any, was Beethoven's third style influenced by his deafness?

55. It is known that Mozart's auricle was abnormally formed. Who wrote a study entitled Mozart's Ohr (1901)? Translate this paper into English.

56. Write on: Robert Schumann (1810-1856), and the Note A which constantly sounded in his ears during his nervous prostration which culminated in tragedy.

57. Discuss the deafness of the German song writer, Robert Franz (1815-1892), and discuss the merits of three of his biographers.

58. What contributions were made to the physiology of the hearing organ during the period 1800-1850?

59. Compile a check-list of periodicals on otology founded during the nineteenth century.

60. What otologists have investigated the hearing power of the aged? What is the presbycusic law?

61. Draw a map of the world, showing where centers of otology were established by the international pupils of Adam Politzer.

62. Write an essay on Russian Otology under the Old Regime, with brief references to the leading workers in St. Petersburg, Moscow, Kiev, Odessa, Kharkov, Riga and Dorpat.

63. Adam Politzer dedicated one of his books (The anatomical and histological dissection of the human ear in the normal and diseased condition, Stuttgart, 1889) to the memory of Albert Burckhardt-Merian (1843-1886). Write a brief study, showing why this tribute was deserved, on: The Position of Albert Burckhardt-Merian in Swiss Otology.

64. Henry Jones Shrapnell, English anatomist of the nineteenth century, is famous for his description of the pars flaccida of the membrana tympani (Shrapnell's membrane), but of the man himself nothing is known. There is no record of his birth or death, and the Germans claim him as their countryman under the name of Otto Shrapnell. If you were assigned the task of writing a sketch of the life and work of Henry Jones Shrapnell, how would you locate his original paper? What would be your procedure in seeking biographical data? How would you know whether a photograph was available?

65. Write on: Hans Wilhelm Meyer (1824-1895), father of otology in Denmark, and benefactor of childhood in all nations.

66. Write on: Felix Delstanche (1802-1892) and Charles Delstanche (1840-1900), builders of Belgian otology, with a survey of the chief Belgian contributions since 1900.

67. Give references to the earliest authenticated cases in which playfully tickling the ear with a stick or other object, or introducing a foreign body in the ear as a practical joke, has resulted in death.

68. Make a list of valuable hearing-aids, and another list of fraudulent hearing-aids.

69. Assume you are an aural surgeon in 1890, and are confronted with the following case: a patient is admitted to your service in the hospital, complaining of pain in the head, nausea, and dizziness; he has a purulent discharge from the right ear, facial paralysis on the right side, and edema of the right optic nerve; his gait is staggering, and you observe his tendency to fall toward the left; you expose the sigmoid sinus and trephine the skull to investigate the temporo-sphenoidal lobe and the upper wall of the tympanic cavity. At the autopsy, there is found in the right cerebellar hemisphere an abscess secondary to chronic suppurative otitis media. Now assume you are confronted with such a case in 1940. What has been learned about this condition in the intervening fifty years?

70. Why have the following names of the ninetenth century become eponyms in otology: William Robert Wills Wilde (1810-1896)? Joseph Toynbee (1815-1866)? Heinrich Adolf Rinne (1819-1868)? Friedrich Eduard Rudolf Voltolini (1819-1889)? Josef Gruber (1827-1900)? Jacob Gottstein (1832-1895)? Emile Siegle (1833-1900)? Robert Robertovich Wreden (1837-1893)? Alexander Prussak (1839-1897)? Dagobert Schwabach (1846-1920)?

71. Write on: Otology's debt to Helmholtz, with a chronology of his contributions to the subject.

72. Write on: Otology in Prague — the work of Emanuel Zaufal (1837-1910), with an addendum on the Czech physicians associated with his clinic.

73. Write on Solomon Moos (1831-1895): his influence on otology in Heidelberg, with sketches of his successors.

74. Write an essay on: The position of Hermann Schwartze in otology, with special reference to mastoidectomy.

75. Write on: Heinrich Neumann (1873-1939), of Hétárs, Hungary, successor to Adam Politzer at Vienna after Victor Urbantschitsch, post-graduate teacher of American physicians, otologist to European royalty, and victim of an Austrian criminal.

76. Trace the principle of binaural listening, as expounded by John William Strutt Rayleigh, from its extension to the use of artificial ears to its application in the detection and location of aeroplanes and submarines. With what name did Lord Rayleigh sign his early papers in physics? Where did he describe his experiments on resonance, limit of audibility, and direction of sound?

77. Who first called attention to bass deafness? boilermaker's deafness? hysterical deafness? labyrinthine deafness? malarial deafness? mental deafness? paradoxic deafness? pocket handkerchief deafness? soul deafness? throat deafness? tone deafness? toxic deafness? vascular deafness? word deafness?

78. In the nomenclature of 1900, the Os temporis, which contains the apparatus of hearing, was divided into a squamous portion, petrous portion, and mastoid portion. Who investigated the development of each of these portions? When was it known that the mastoid process is not present at birth, but appears about the second year and becomes pneumatic about puberty? What are the authoritative texts on the embryology of the temporal bone?

79. Who was the first otologist to receive the Nobel prize? Give a brief summary of his epochal work.

80. When was the liquor of Karl August von Burow (Burow's solution) first used for a boil in the external auditory canal? Who suggested warm instillations of absolute alcohol for otomycosis? Who first recommended phosphorus and cod liver oil in otosclerosis? Who was the first to employ boric acid in chronic otorrhea? Who introduced the vaccine treatment of suppurative otitis media? Who was the first to instill a solution of cocaine into the ear? In the radical mastoid operation, who first packed the cavity with iodoformized gauze? Who first used blood serum of convalescing cases of epidemic parotitis as a preventive of this condition?

81. A Festschrift was presented to Ino Kubo on his sixtieth birthday (December 26, 1934). State what you would have written for this Festschrift if you had been invited to contribute the introduction, on: The position of Ino Kubo of Fukuoka in Japanese otology.

82. Who was the first American otologist? Who wrote the first American treatise on otology? Name the first American teachers of otology.

83. Write a review of the following American pamphlets and memoirs: Samuel Latham Mitchill: A discourse pronounced by request of the Society for Instructing the Deaf and Dumb (New York, 1818, 32 pages); Samuel Gridley Howe: Remarks upon the education of deaf-mutes, in defense of the doctrines of the second annual report of the Massachusetts Board of State Charities, and in reply to the charges of the Rev. Collins Stone, principal of the American Asylum at Hartford (Boston, 1866, 58 pages); Gardiner Greene Hubbard: The education of deaf-mutes. Shall it be by signs or articulation? (Boston, 1867, 36 pages); Alexander Graham Bell: Memoir upon the formation of a deaf variety of the human race (Washington, 1883-1885, 86 pages, quarto); Edward Allen Fay: Marriages of the deaf in America (Volta Bureau, 1898, 527 pages).

84. Write on: The Gallaudets, an American epic — Thomas Hopkins Gallaudet (1787-1851) and the first institution for deaf-mutes in America; Thomas Gallaudet (1822-1902) and the first church for deaf-mutes in America; Edward Miner Gallaudet (1837-1917) and the first college for deaf-mutes in America. Why do the wives of the Gallaudets add considerably to the interest of the story?

85. Oliver Wendell Holmes (Border Lines in Medical Science, 1861) said: "It were to be wished that the elaborate and very interesting researches of the Marquis Corti, which have revealed such singular complexity of structure in the cochlea of the ear, had done more to clear up its doubtful physiology; but I am afraid we have nothing but hypotheses for the special part it plays in the act of hearing, and that we must say the same respecting the office of the semicircular canals." What progress, if any, has been made in this field since that time?

86. What otologists were hard of hearing, or suffered from increasing deafness with advancing years? What celebrated educators of the deaf married deaf girls?

87. To serve as a chapter of a symposium on the Melting-pot in Medicine, write on Foreign pioneers of otology in California, who settled in San Francisco about 1870: Narcisse-Joseph Martinache (1833-1892) of Picardy; Henry Ferrer (1850-1890) of Cuba; Adolph Barkan (1845-1935) of Hungary; with a list of foreign-born physicians in America who specialized in diseases of the ear.

88. The following were deaf or extremely hard of hearing: the inventor of the phonograph (1877); the newspaperman who composed the baseball classic, Casey at the Bat (1888), of which it is said that ten thousand impostors claimed the authorship; and the physiologist who wrote The Elements of the Science of Nutrition (1906). Identify these Americans, and prepare an article suitable for a magazine of general circulation on: Defeating the Disability of Deafness — the American way of overcoming an ancient handicap.

89. Who wrote the section on Otology in the United States for the second volume of Politzer's Geschichte der Ohrenheilkunde? With what portraits of American otologists is it illustrated? To what American otologists is this volume dedicated?

90. In what year and by whom were the following American periodicals established: American Annals of the Deaf and Dumb? American Journal of Otology? Annals of Otology, Rhinology, and Laryngology? Archives of Otolaryngology? Archives of Otology? Journal of Eye, Ear, Nose, and Throat Diseases? Journal of Ophthalmology, Otology, and Laryngology? The Laryngoscope? Volta Review?

91. What otologists are included in: (a) Dictionary of National Biography? (b) Dictionary of American Biography? If you were the editor of these dictionaries. what names which have been omitted would you have included?

92. What school for the deaf was designed and built under the direction of a deaf architect? Illustrate your paper with the blue-prints.

93. Discuss the relative merits of the American textbooks on otology by Laurence Turnbull (1872), Daniel Bennett

Saint John Roosa (1873), Charles Henry Burnett (1877), and Albert Henry Buck (1880).

94. "Scarlet fever in children is the most frequent cause of permanent deafness and deaf-mutism" (Gorham Bacon). — Who wrote the original account of scarlatina? Who wrote the classic account? At what period did it become known that the infection of scarlet fever may pass from the nose and throat to the Eustachian tube and fill the middle and inner ears with pus? What important discoveries in scarlatina have been made by the Chicago physicians, George Frederick Dick and Gladys Rowena Henry Dick?

95. What is the otologic interest in Clarence Darrow's The Story of My Life (1932)? What other non-medical autobiographies contain references to diseases of the ear?

96. Write a true story for children on: Edward Bartlett Nitchie (1876-1917), the deaf boy who won a Phi Beta Kappa key; how he looked for a job and learned that employers did not want an employee who could not hear; how he studied lip-reading and opened a school for those afflicted as himself; how he courted his sweetheart over the proofs of his first book, and wrote standard volumes on lip-reading; how he edited the little magazine, Courage; how he lectured to the deaf in various cities on English literature, and taught others to do so; how he was invited to address the Section on Otology of the New York Academy of Medicine; how he inaugurated the New York League for the Hard of Hearing; how he established free lip-reading scholarships, with which the name of Annetta Peck is associated; how he attempted to introduce a correspondence-course for the deaf; how he trained Juliet Clark, Jane Walker, and Pauline Ralli to become efficient instructors of his method; how he struggled against increasing ill-health, and worked for his school even when he was dying; how his wife, true to her promise, conducted the school for ten years until her own health failed; and how the school finally was incorporated by the Board of Regents of New York State.

97. Discuss: The teaching of lip-reading as a career for superior women; with references to the American pio-

neers, Sarah Fuller, Harriet Rogers, Mary True; and the later workers, Sarah Warren Keeler, Alice Jennings, Lillie Eginton Warren, Sarah Allen Jordan, Martha Bruhn, and the sisters Cora Elsie and Rose Kinzie.

98. Write on: Origin and development of lip-reading in public school evening classes, with special reference to the work of Virginia Osborn (Cincinnati); Katherine Ashelby (Chicago); Mary Woodrow (Brooklyn); Louise Morgenstern, Estelle Samuelseon, Ida Becker (Manhattan); Sally Tripp (Boston); Enfield Joiner, Juliet Clark, Jane Walker, Martha Bruhn (lip-reading for deafened soldiers).

99. Prepare a pageant illustrating the main events in the careers of the three generations of the Bell family: Alexander Bell (1790-1865), Alexander Melville Bell (1819-1905), Alexander Graham Bell (1847-1922). The pageant is to bring out the significance of this family in the story of speech and the education of the deaf.

100. Prepare a pageant on The Deaf, depicting their degraded status before the era of education, the first private schools for the instruction of the few paying deaf, the democratic movement for the compulsory education of all the deaf, concluding with a panorama of several celebrated men and women who were deaf.

Temple University School of Medicine.

ENDAURAL FENESTRATION OF THE HORIZONTAL SEMICIRCULAR CANAL FOR OTOSCLEROSIS. INDICATIONS, TECHNIQUE, OBSERVATIONS AS TO EARLY AND LATE POST-OPERATIVE RESULTS.*

DR. JULIUS LEMPERT, New York.

That hearing could be temporarily improved in otosclerosis by fistulization of the labyrinth was known and reported for very many years, but the permanent restoration of serviceable physiological hearing by air conduction in otosclerosis was awaiting the development of a surgical technique. Any technique which fails to permanently restore the hearing to the decibel threshold necessary for serviceable conversational hearing is of no social or economic value to the patient and should, therefore, be of no interest to the otologist. The endaural one-stage fenestration technique[1] which I employ and advocate for the surgical treatment of otosclerosis results in a permanent restoration of practical hearing by air conduction which can always be substantiated audiometrically and by all other scientific means.

INDICATIONS.

Fenestration of the external semicircular canal is indicated:

1. When the hearing loss is bilateral and progressive.

2. When the stapes within the fenestra ovalis is fixed either as a result of ossification of the annular ligament or an ankylosis of the incudomalleolar joint.

3. When the round window membrane has remained normal.

4. When the Eustachian tube is patent.

5. When the tympanic membrane is normal and completely intact. A complete absence of middle ear infection is abso-

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lutely essential, and a history of never having had a middle ear infection before is preferable.

6. When the hearing by air conduction in the conversational frequencies 512, 1,024 and 2,048 has receded below the threshold necessary for serviceable conversational hearing.

7. When the hearing by bone conduction for the frequencies 512, 1,024 and 2,048, as determined audiometrically under the condition of masking, has remained normal or receded to a level not lower than 30 dcb. Bone conduction is the index of cochlear nerve function; therefore, when the bone conduction in the conversational frequencies has receded to a level below the above indicated levels for normalcy, fenestration is contra-indicated.

8. A normal state of health is essential.

Our diagnostic means for determining which cases are definitely suffering from otosclerosis are rather meagre. I am, however, thoroughly convinced that otosclerosis is much more prevalent than it is generally assumed to be, and that many cases diagnosed and labeled as otitis media catarrhalis chronica are really cases of otosclerosis.

A permanent restoration of the highest degree of practical physiological hearing by air conduction can be obtained following this fenestration technique in any patient suffering from a loss of air conduction hearing, no matter how great, in the presence of the aforementioned requirements.

The indications described were arrived at in retrospect after a careful observation and study of many cases operated upon without a clear understanding of what was actually required in order to obtain the permanent restoration of practical hearing following fenestration of the labyrinth.

The following methods were employed to determine the preoperative and postoperative hearing state:

1. Audiometric testing which included an air conduction determination, a bone conduction estimation, and a bone conduction estimation under the condition of masking. A 6A Western Electric audiometer was employed for these tests.

2. Fork tests both by air and bone conduction.

3. Testing by normal conversation and whisper.

The hearing in each case was tested two to three times at different intervals before operation, and each month postoperatively.

SURGICAL ACCIDENTS.

1. Fracture of the vertical mastoid portion of the Fallopian canal: In removing the inferoposterior bony canal wall down to the level of the vertical portion of the facial canal, the bony capsule of the Fallopian canal was accidentally fractured and the facial nerve was exposed but not injured. This resulted in facial nerve paresis, which gradually subsided. The bony mastoid structure in this case was exceptionally brittle.

2. In two cases, where an extremely contracted mastoid process was encountered, with the lateral sinus impinging upon the posterior bony wall of the external auditory canal, the sinus was accidentally punctured. The bleeding was easily controlled. The operation was successfully completed. The postoperative course and convalescence remained uncomplicated.

POSTOPERATIVE SEQUELLAE.

1. Two cases developed a serous labyrinthitis two to three days postoperatively. This was apparently due to a severe inflammation of the tympanomeatal membrane extending to the perilymph space. In these two cases, the serous labyrinthitis resulted in a complete loss of vestibular and cochlear function.

2. Two patients died of coronary thrombosis. Permission for autopsy in one of these cases was obtained. The diagnosis of thombosis of the right circumflex branch of the coronary was confirmed. The temporal bone, together with the entire tympanomeatal membrane covering the fenestra in the external semicircular canal, was removed intact for microscopic study by Dr. Joseph Druss.

These two deaths were purely coincidental and had no relation whatsoever to the operation.

POSTOPERATIVE INSPECTION AND REVISION OF THE FENESTRA IN THE EXTERNAL SEMICIRCULAR CANAL.

I have investigated and revised postoperatively the newly created fenestra in the horizontal canal in 39 of the 150 cases operated upon, in order to determine the cause for the failure to maintain permanently the hearing improvement which was obtained temporarily by fenestration in these cases. In four of these cases, the fenestra was revised twice, and in one case four times, thus 46 revisions were performed on the 39 fenestrae.

Twenty-three cases were revised because the hearing improvement in these cases receded to the preoperative level within a period of six to eight weeks postoperatively. The vestibular response to the fistula test, which gradually diminished in its activity became completely negative, while the vestibular function of the membranous labyrinth remained normal. In 21 of those cases, fibrosis was found to be the cause of closure. Six cases were subjected to inspection and revision of the fenestra because the improvement in hearing which was obtained by the fenestration receded to the preoperative level several months later, in spite of the fact that the vestibular response to the fistula test remained permanently positive. The activity of the vestibular response, however, in these cases was considerably diminished as compared to the activity of the response obtained the first few postoperative weeks. In every one of these cases, circumscribed osteogenesis was found to have decreased the size of the fenestra without resulting in complete closure of the fenestra. Two cases were inspected and revised because the fenestra was originally made inadequate in size, and as soon as the fenestra was covered with the tympanomeatal membrane, the hearing improvement obtained on the table receded to the preoperative level and remained so permanently. The vestibular response to the fistula test was positive in these cases. Inspection of the fenestra revealed the fact that no new bone regeneration had taken place and confirmed the suspicion that the failure to improve hearing was due to the inadequacy of the size of the fenestra. Seven cases were subjected to inspection and revision of the fenestra because the hearing improvement obtained by fenestration receded to the preoperative level soon after operation, while the vestibular

response to the fistula test, which was very active for the first few weeks, suddenly became very sluggish and protracted. No new bone regeneration was observed in these cases. The membranous labyrinth was found to be inflamed, thickened and bound by adhesions to the endosteum. One case was inspected and revised because, following the original operation, the tympanomeatal membrane sloughed away, leaving the fenestra completely exposed and uncovered. The hearing improvement obtained on the table receded quickly postoperatively and gradually reached a level much lower than the preoperative level of hearing. The response to the fistula test became rapidly negative and the vestibular function became gradually but increasingly impaired. The findings in this case consisted of a fan-shaped band of fibrous tissue beginning on the surface of the bony structure of the floor of the mastoid cavity in the region below the posterior end of the horizontal semicircular canal and extending anteriorily and upwards into the fenestra and finally into the perilymph space, compressing and displacing the membranous labyrinth to which it became attached.

SUMMARY REPORT OF HEARING IMPROVEMENT RESULTS.

The vast majority of the patients operated upon were first examined and then referred to me by otologists of the highest standing. Those patients who applied to me directly for surgical relief of their deafness were referred by me for examination to an otologist of their own choice in their own community, prior to my consenting to examine them personally in order to determine whether they were suitable for operation. Every one of the patients operated upon was treated for deafness unsuccessfully by many otologists for many years prior to submitting to the fenestration operation.

The fenestration operation was performed in 150 cases within the past two and one-half years.

A permanent restoration of practical physiological hearing resulted in 93 cases. These 93 patients are now socially and economically rehabilitated.

A marked improvement in conversational hearing resulted in 11 cases. The improvement of hearing in these 11 cases, though audiometrically impressive, did not, however, reach

the level of improvement necessary for the restoration of practical hearing.

A further impairment of hearing resulted in 14 cases. The impairment of hearing in these cases, though audiometrically significant, practically, however, did not in any way affect these patients, as none of them were able to hear practical conversation prior to surgical intervention.

The hearing remained unimproved in 32 cases.

All my good hearing improvement results, including all my failures, were constantly observed and repeatedly seen for the past two and one-half years by most of the leading otologists of this country and many otologists from other countries.

In every case where a permanent restoration of practical physiological hearing was obtained following fenestration, tinnitus completely disappeared on the operative side. In those cases where the hearing improvement did not reach the practical level, the intensity of the tinnitus was greatly diminished. In those cases where the hearing remained unimproved the tinnitus remained the same. In those cases where the hearing was further impaired following the fenestration, the tinnitus was proportionately intensified. This seems to point to the possibility that a direct relationship exists between the loss of hearing and the tinnitus in otosclerosis.

Although the fenestra in the external semicircular canal remained permanently open in 126 of the 150 cases operated upon, the hearing was improved in only 104 of these cases. Thus, in 21 of the 126 cases, where the newly created fenestra had not closed, the hearing, nevertheless, remained unimproved.

The improvement in hearing resulting from the fenestration of the horizontal semicircular canal is one of air conduction only.

The hearing by bone conduction is neither improved nor impaired by a successfully performed fenestration. It remains unchanged.

In all the cases wherein the fenestra closed, such closure was usually complete within a period of three to 10 weeks

postoperatively. When the fenestra did not show signs of beginning closure by a diminution in the activity of the vestibular response to the fistula test at the end of eight weeks postoperatively, the fenestra remained permanently open thereafter.

In none of the cases operated upon did I ever observe a practical hearing improvement which could be audiometrically substantiated in the nonoperated ear. Since the improvement in the operated ear is one of air conduction only and not bone conduction, the improvement in hearing following fenestration cannot be interpreted as an improvement in the central perception of sound. We cannot improve the air conduction hearing in both ears by reconstructing the air conduction mechanism in only one ear. Fistulization of the labyrinth does not improve the function of the cochlear nerve but, instead, it improves the function of the air conduction mechanism for the transmission of air-borne sound to the cochlear nerve.

When the fenestration procedure suggested by me was performed in the presence of good bone conduction and before secondary nerve changes had taken place in the three conversational frequencies, 512, 1,024 and 2,048, the restoration of practical hearing was obtained and permanently maintained in 80 per cent of the cases.

The following audiograms will serve as an example of the type of hearing improvement obtained in the successfully operated cases.

ANALYSIS OF HEARING IMPROVEMENT RESULTS OBTAINABLE FOLLOWING OTOSCLEROSIS SURGERY.

Hearing improvement results may be classified as follows:

1. Immediate Hearing Improvement Results: Any kind of a fistula, large or small, made in any part of any one of the semicircular canals, with either a sharp or dull instrument, with or without injuring the membranous labyrinth, will result in an immediate improvement of hearing of the highest obtainable degree; however, a hearing improvement obtained in this manner, although very spectacular in the operating room, cannot be subsequently maintained.

2. Early Temporary Hearing Improvement Results: A fistula created within the bony capsule of any one or every one of the semicircular canals, through either the endaural, postauricular or the combined endaural-postauricular routes,

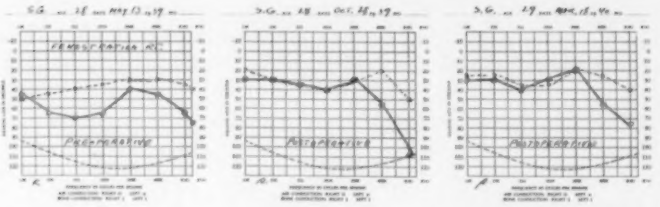

Fig. 1.

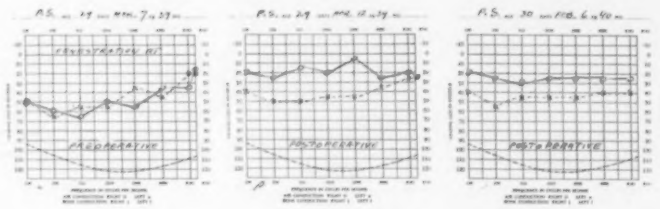

Fig. 2.

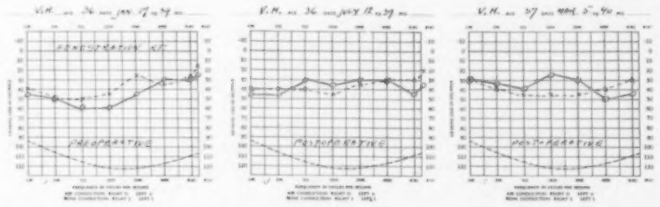

Fig. 3.

when covered by any kind of a membrane and surrounded by an air space, will result in an improvement of hearing of varying degree. This improvement may last from two weeks to two months, providing the membranous labyrinth has not been destroyed during the process of fenestration and a diffuse serous labyrinthitis has not occurred postoperatively.

3. Late Permanent Hearing Improvement Results: Practical serviceable hearing for air-borne sound will be restored and permanently maintained when: *1.* The surgery for otosclerosis is based on the theory that there exists in otosclerosis an impedance to the mobility of the perilymph and

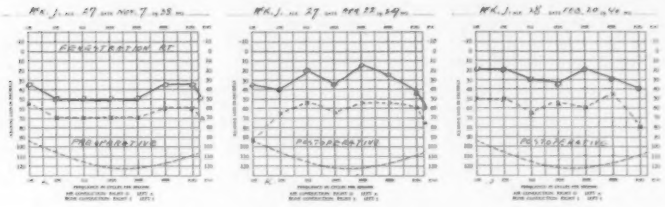

Fig. 4.

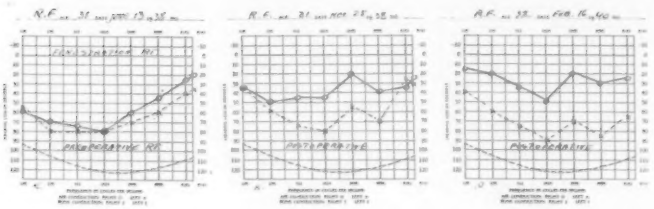

Fig. 5.

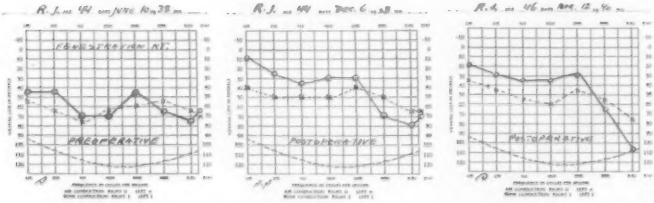

Fig. 6.

endolymph for air-borne sound. *2.* Every step in this surgery is planned and executed so as to overcome the existing impedance in order to re-establish the degree of perilymph mobility necessary for the restoration of serviceable physiological air conduction hearing. *3.* The technique employed provided ways

and means for maintaining permanently the newly established unimpeded perilymph mobility for air-borne sound by forestalling and preventing some new form of impedance to the perilymph mobility from manifesting itself postoperatively.

It has been known and observed for very many years that when a fistula is made in the vestibular portion of the bony capsule of the labyrinth in an otosclerotic ear, before cochlear

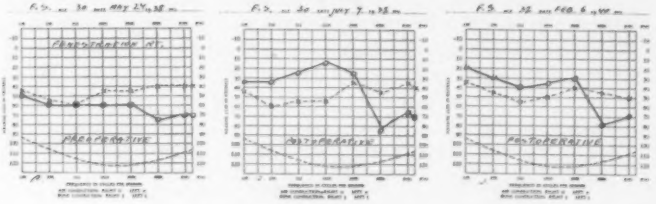

Fig. 7.

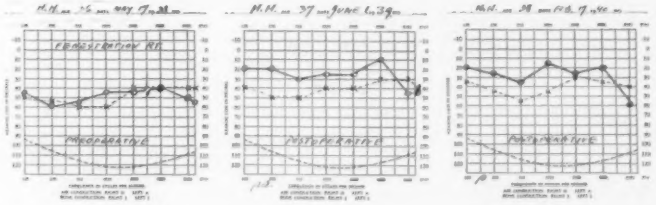

Fig. 8.

nerve degeneration has taken place in the conversational frequencies, there occurs an immediate improvement of hearing for air-borne sound of the highest obtainable degree in that patient. Most otologists who observed this phenomenon, and some who occupied themselves with the development of the surgery for otosclerosis, have always believed that the improvement of hearing resulting from such a fistula was the direct result of the decompression of a supposedly existing, but never proven, increased intralabyrinthine fluid pressure. Despite their observation that the hearing improvement could be temporarily maintained after such a fistula was covered and sealed tightly with a membrane, thus permitting

the perilymph to refill the perilymph space, they still adhered to the theory of decompression. Holmgren[2] argues that there exists the possibility of the perilymph being resorbed through the membrane covering the fistula. Sourdille[3] advanced a half-bottle theory to support the theory of decompression.

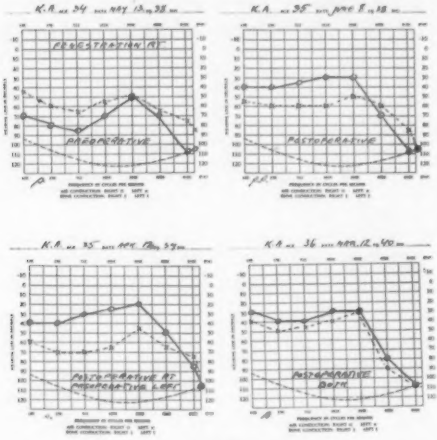

Fig. 9.

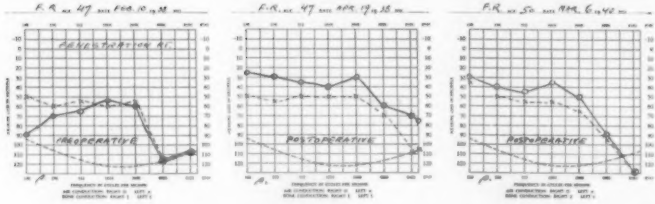

Fig. 10.

My own observations have failed to reveal any facts which could help support this theory. On the contrary, they all pointed away from it. At no time was I able to observe an escape of perilymph into the mastoid wound which could compare to the escape of spinal fluid following the minutest opening of the subarachnoid space. I never saw the perilymph reach beyond the level of the lumen of the semicircular canal,

though I have patiently watched for it for periods of as long as two hours, thus convincing myself that the perilymph was never under increased pressure in these cases. By removing the perilymph and watching for it to refill the perilymph space, the slowness with which the perilymph entered the perilymph space led me to suspect that there must exist a natural barrier between the cerebrospinal fluid system and the perilymph space which controls the flow of perilymph and permits only as much perilymph to get through as is necessary to cushion the membranous labyrinth with its endolymph, and enough to maintain the necessary amount of pressure relationship between the perilymph and endolymph, but no more. Whereas, perhaps in and under certain conditions the amount of endolymph may be increased, this is always at the expense of the perilymph space, which then permits a lesser amount of perilymph to enter and cushion the endolymph. Thus a change in normal pressure relationship between the perilymph and endolymph may occur, but no increased perilymph pressure within the lumen of the canal was ever observed to exist.

If the decompression theory had a basis in fact, a fistula in the cochlear promontory should result in improved hearing, but it never does. Neither does the creation of a new window in the external semicircular canal improve hearing when either the round window alone or both the oval and round windows are functionally impeded. Whereas a new window created in the external semicircular canal in the presence of a functionally impeded oval window does improve hearing by air conduction, if the round window membrane is normal. Since two functionally unimpeded windows, one in the vestibular part and another in the cochlear part of the bony labyrinthine capsule, are essential to improve air conduction hearing, and since it is possible to maintain the hearing improvement obtained by fistulization after covering and sealing the fistula with a membrane, I am convinced that the hearing improvement obtained following fistulization of the external semicircular canal is not the result of decompression of intralabyrinthine fluid pressure but rather the result of the mobilization of the perilymph and endolymph by airborne sound, which was heretofore hindered by the functionally impeded oval window, and which the newly created

window in the external semicircular canal again makes possible. I believe that the reason the hearing improvement which immediately follows the opening of the perilymph space is of the highest obtainable degree is because an unnatural state is created, permitting air-borne sound to mobilize the perilymph by coming in direct contact with it.

Based upon this theory of impedance, the following technical requirements for the permanent re-establishment of a free mobility of the perilymph for air-borne sound are essential in order to obtain late permanent hearing improvement results in otosclerosis:

1. The creation of a fenestra in the external semicircular canal to mobilize the perilymph for air-borne sound.

2. The covering of the newly created fenestra with a membrane to prevent the membranous labyrinth from being damaged by either exposure or infection.

3. The anatomical reconstruction of the tympanic air space to serve best physiologically the newly created fenestra.

4. The employment of a technique for the achievement of these technical requirements which will result in a minimum of postoperative inflammatory reaction of tissues.

HOW SHOULD THE FENESTRA BE MADE IN ORDER TO OBTAIN PERMANENT LATE RESULTS?

An architecturally planned fenestra instead of any kind of a fistula should be created in the external semicircular canal to replace the functionally impeded fenestra ovalis. Holmgren[2] stated as follows: "How shall the fistulae be made in order to produce the greatest primary improvement in hearing, and how shall they later be handled in order to achieve the greatest permanent good? Those are the two important questions demanding attention. Once these are answered, one may expect that the surgical management of otosclerosis will be successful in the hands of those who can maintain the necessary asepsis."

The best location for the creation of the fenestra is the external semicircular canal, because a fenestra in this region is nearest to the functionally impeded oval window and can, therefore, be readily enclosed within, and made part of a

reconstructed tympanic cavity which is covered with the tympanic portion of the tympanomeatal membrane. *An adequate* fenestra should measure 7 mm. to 9 mm. in length and 1.5 mm. to 2 mm. in width, so that when covered with the membrane, the impedance to air-borne sound created by the membrane could be overcome by the size of the fenestra and thus result in the amount of free mobility of the perilymph and endolymph for air-borne sound necessary for the restoration of practical hearing. The width of the fenestra should represent a semicircle of the circumference of the lumen of the canal obtained by the removal of the outer and posterior walls of the bony capsule of the external semicircular canal. That a fenestra, in order to be adequate and result in practical hearing after being covered, must necessarily be of the dimensions described, has been observed by creating fenestrae varying in size from 2 to 9 mm. in length and from 1 to 2 mm. in width; furthermore, I have observed that whenever a fenestra, originally made adequate, began to diminish in size because of new bone regeneration, as shown by the gradually diminishing activity of the vestibular response to the fistula test, the hearing soon receded to the preoperative level. Inspection and revision of cases where a diminution of the size of the fenestra was suspected to be the cause of the recession in hearing revealed the presence of a circumscribed area of osteogenesis which diminished the diameter of the fenestra but was not sufficient to close it.

A small fenestra when covered with a membrane becomes sufficiently impeded to prevent the establishment of the degree of perilymph mobility necessary for the restoration of serviceable hearing by air conduction.

The depth of the fenestra should extend from the outer surface of the bony capsule through the bony capsule and through the endosteum lining its lumen, and should reach and communicate with the perilymph space. *The endosteum must be removed.* The removal of the endosteum must be accomplished without injuring the membranous labyrinth. Since the membranous labyrinth occupies a position within the lumen of the canal closest to the convexity and furthest away from the concavity of the canal, the removal of the endosteum should be begun in the posterior wall of the canal in the region of the concavity where the perilymph space is widest,

in order to reduce to a minimum the risk of injuring the membranous labyrinth.

The removal of the endosteal membrane without injury to the membranous labyrinth is an art which is accomplished successfully only after extensive training and experience. One must first learn how to visualize and recognize the membranous labyrinth *in vivo* and then how to distinguish the endosteal membrane from the membranous labyrinth.

The appearance of a small drop of perilymph through a microscopic perforation in the endosteum bathing the outer exposed surface of the endosteum will often mislead the inexperienced surgeon into believing that he has exposed the perilymph space and, as a result, into mistaking the endosteal membrane for the membranous labyrinth. Beginners in this type of surgery can be classified as belonging invariably to one of two groups: *1*. Those who, without proper training and without appreciation of the surgical requirements for successful fenestration of the labyrinth, proceed to make a fistula instead of the required fenestra, without regard for the safety of the membranous labyrinth. Their results are negative or worse. They usually perforate both the endosteum and the membranous labyrinth. *2*. Those who, after extensive training, acquire such a respect and reverence for the delicacy of this surgery that they approach the act of fenestration with such trepidation, for fear of injuring the labyrinth, that they leave the endosteal membrane intact and seldom succeed in exposing the membranous labyrinth. Their results are often good, but not lasting.

That the endosteum must be removed to the limits of, and if possible beyond, the edges of the fenestra has been concluded from the following observations:

1. Though the hearing improvement following immediately upon exposure of the endosteum is quite impressive, when such a fenestra is covered with a membrane, the hearing improvement seldom reaches the level necessary for practical hearing. The reason for this is most likely the fact that the amount of impedance to the mobility of the perilymph created by the endosteum plus the membrane employed to cover the fenestra, though not sufficient to prevent improvement in hearing, is sufficient, however, to prevent the restoration of practical hearing.

2. The endosteum left intact is generally one of the most responsible factors for the closure of the fenestra by osteogenesis. Whenever a fenestra was inspected soon after it became evident that the fenestra closed as a result of new bone regeneration, an endosteal lid of newly formed bone, about 0.1 mm. in thickness, was inevitably found. No new bone regeneration was ever observed in the periosteal layer of the walls of the fenestra in any of the early revisions. When a fenestra was revised many months after evidence of its closure by osteogenesis existed, both the periosteal and endosteal layers were found to be regenerated. This would suggest that osteogenesis begins in the endosteum. The enchondral layer was never observed to have regenerated.

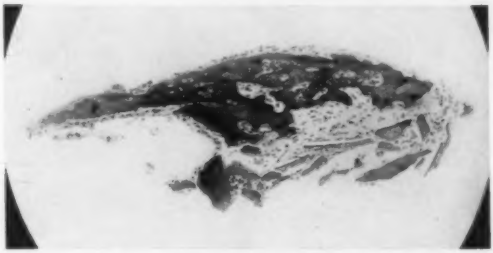

Fig. 11. Microphotograph of newly formed endosteal bony lid.

The careful removal, therefore, of the endosteum is one of the important factors in obtaining late permanent hearing improvement results.

Every possible precaution must be taken not to injure the membranous labyrinth. Surgical injury of the membranous labyrinth with escape of endolymph usually results in further impairment of hearing.

The superoanterior and inferoposterior bony margins of the longitudinal diameter of the fenestra should be rounded off to equal in width the widest diameter of the fenestra. The upper and lower ends of the fenestra should *never be permitted* to be triangular in shape, for such a condition will only facilitate new bone regeneration.

All microscopic particles of bone dust and bone chips must be removed from the lateral bony walls of the fenestra, from

the cut edges of the endosteal membrane and from the perilymph space. Failure to completely remove bone dust from the perilymph space will result in formation of new bone in this space and this will result not only in a recession of the the hearing improvement but in a still further impairment of hearing.

Fig. 12 (colored plate). Newly formed bone within the perilymph space resulting from bone dust.

Fig. 13. Microphotograph of newly formed bony mass removed from perilymph space resulting from bone dust.

The perilymph space should never be opened in the presence of even the most infinitesimal bleeding. Every precaution must be taken to avoid blood from accumulating in the fenestra and entering the perilymph space. When blood is permitted to enter the perilymph space, fibrous tissue forms and closes the fenestra. The hearing in such a case usually

becomes still further impaired. Inspection and revision of the fenestra in such a case revealed a mass of fibrous tissue obliterating the new fenestra and extending into the lumen of the external semicircular canal.

Such an ingrowth of fibrous tissue cannot be removed without injuring the membranous labyrinth to which it is usually attached.

It can readily be seen and understood that success or failure of surgery for otosclerosis will depend upon the degree of skill and art employed in the creation of the fenestra in the external semicircular canal; therefore, just as long as one of the surgical requirements for the improvement of hearing in otosclerosis will be the creation of a fenestra, the results will always vary with the skill, surgical temperament and experience of the surgeon.

The fenestration of the bony walls of the external semicircular canal is best begun with a dental polishing burr and completed with a gold burnishing burr. Such treatment of the bony walls of the fenestra diminishes the power of periosteal bone regeneration but does not stop it. I have always disagreed with the conclusions drawn and published by Canfield,[4] which were based on the results of his investigation of the effect of the polishing and burnishing burrs upon osteogenesis. After making various defects on the skull of a cat, some with sharp cutting instruments and others with dull polishing and burnishing burrs, and covering these defects with the periosteum-lined scalp, Canfield found that after two weeks new bone regeneration had taken place in all the defects made, with the exception of the defects made with the polishing and burnishing burrs, which showed no new bone regeneration. The publication of the results of this experiment created the impression that the polishing and burnishing burrs were solely responsible for the prevention of new bone regeneration. Because of my extensive experience with the use of these burrs, and my opportunities to observe the effects of employing them for several years, I am convinced that my success in maintaining the fenestra permanently open in the vast majority of my cases was due not alone to the use of these burrs, but that other factors in my technique, plus the use of these burrs, were together responsible for the prevention of osteogenesis. Actually, a

number of my cases did result in osteogenesis in spite of the fact that I made the fenestra with polishing and burnishing burrs. If the burr were the sole factor, why would osteogenesis take place in some and not in others? I believe that the heat produced by the use of these dull burrs helped to prevent osteogenesis in my cases by partially devitalizing the osteoblasts; however, I am convinced that if I had done nothing more to prevent new bone regeneration, osteogenesis would have eventually taken place, but not as rapidly as if the sharp burrs had been employed.

Strongly believing in my contention, I decided to investigate this phase in the research of the problem of osteogenesis. At my suggestion, various defects were made by Dr. Philip Meltzer, of Boston, in the skulls of two cats, with the aid of sharp burrs, curettes and saws, and the polishing and burnishing burrs, and all defects were covered with the periosteum-lined scalp. One cat was permitted to live two weeks, and one six weeks. Every one of the two-week old and six-week old defects made with sharp instruments showed progressive stages of osteogenesis. The two-week old defects made with the polishing and burnishing burrs showed no signs of new bone regeneration, the same as in Canfield's[4] cat after two weeks; however, the defects made with the polishing and burnishing burrs in the cat which was permitted to live six weeks showed the beginning of new bone regeneration. The microscopic sections were prepared and interpreted by Dr. Spector.

The results obtained with our experiment point to the fact that the polishing and burnishing burrs retard bone regeneration in periosteal bone but do not prevent it.

HOW SHOULD A FENESTRA IN THE EXTERNAL SEMICIRCULAR CANAL BE COVERED AND PROTECTED?

A fenestra created in the external semicircular canal cannot be left exposed to the air without predisposing the membranous labyrinth to damage resulting from exposure or infection. A fenestra must, therefore, be covered and protected with a viable membrane. As soon as the fenestra is covered with a membrane, the marked hearing improvement which immediately followed fenestration recedes. The degree

to which the hearing improvement recedes depends upon the amount of impedance introduced by the membrane to the mobilization of the perilymph by air-borne sound. The degree of impedance resulting from the application of the membrane over the fenestra depends upon the thicknes of the membrane and the manner in which it is applied to cover the fenestra,

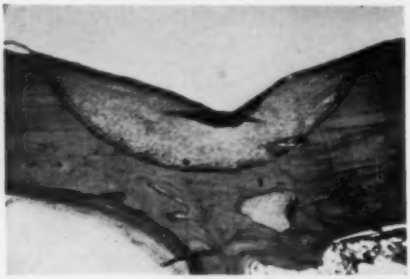

Fig. 14. Cat No. 3, 29 days. Defect made with sharp burr — defect burnished afterward. Defect filled with periosteal tissue. No regeneration of bone.

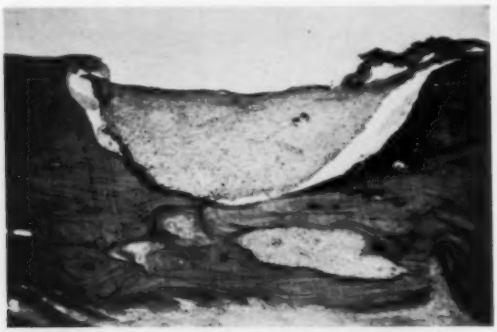

Fig. 15. Cat No. 3. 29 days. Defect made with gold polishing or dull burr. No regeneration of bone.

which can either impede or facilitate the mobility of the membrane for its conduction and transmission of air-borne sound to the perilymph.

In order to least impede the mobility of the perilymph for air-borne sound, the membrane employed to cover the fenestra should be the thinnest which can possibly be obtained and

should be epithelium-lined on both its outer and inner surfaces so that it will not stimulate new bone regeneration

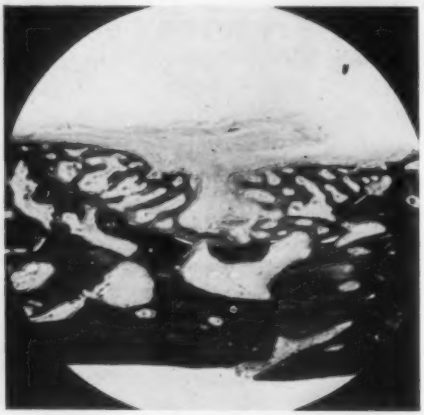

Fig. 16. Cat No. 2, left side, 42 days. Defect made with sharp burr—unburnished. Much more bone regeneration as compared to the right side, where defect was made with sharp burr but defect was burnished.

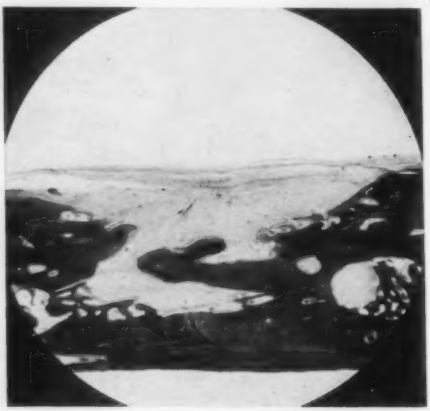

Fig. 17. Cat No. 2, left side, 42 days. Defect made with gold polishing burr, unburnished. Shows very little bone regeneration. Compares well with right side, where gold polishing burr was used, but defect was burnished.

within the bony walls of the fenestra. It is for this reason that I employ Shrapnell's membrane, which has an outer der-

mal surface and an inner epithelium-lined mucosal surface to cover the fenestra. In order to permit Shrapnell's membrane

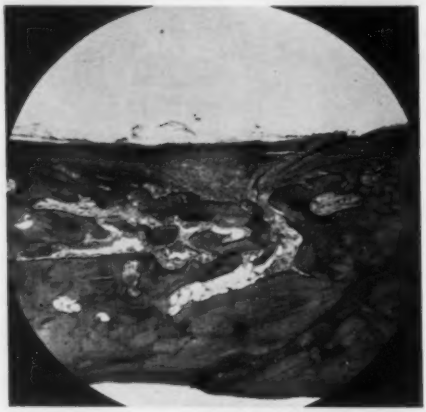

Fig. 18. Cat No. 2, right side, 42 days. Defect made with sharp burr. Defect was then burnished with smooth burr. Regeneration of bone quite marked as compared with defect made by dull polishing burr.

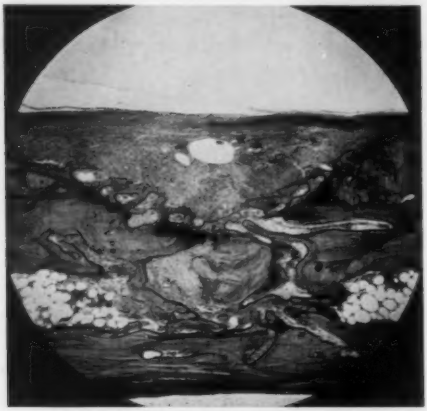

Fig. 19. Cat No. 2, right side, 42 days. Defect made with gold polishing burr. Defect then burnished with smooth burr. Shows some regeneration of bone but much less than defect of same age in same cat when sharp burr was used.

to reach and cover the new fenestra, it is necessary to mobilize and free the entire tympanic membrane from the points

of its fixation; therefore, the head and neck of the malleus must be removed in order to liberate the tympanic membrane from its anchorage to the ossicular chain. The tympanic memberane should also be free from the entire circumference of the sulcus tympanicus, leaving it attached only to the anterior margin thereof. This would lower it and bring it closer to the inner tympanic wall and, through the extension of Shrapnell's membrane backward in a straight line, enable it to reach and cover the fenestra in the external semicircular canal. *Since a straight line is the shortest distance between two points,* the extension of Shrapnell's membrane to the external semicircular canal is made possible. This, however, would be impossible to accomplish if the tympanic membrane were permitted to occupy its normal position within the sulcus tympanicus which does not form a straight line with the external semicircular canal. I strongly believe that the reason why the fenestra remained open in 126 of the 150 cases operated upon is the fact that in addition to the many precautionary measures which I employ to prevent osteogenesis, the employment of the inner epitheliated surface of Shrapnell's membrane to cover the fenestra is a contributory factor of major importance. I also believe that in some cases where I do not succeed in covering the entire fenestra with Shrapnell's membrane, the epithelium grows quickly, spreads and extends from the inner surface of Shrapnell's membrane to the immediately adjacent and attached thin cutaneous membrane for a distance sufficient to cover the rest of the fenestra. *Though a fenestra may occasionally remain permanently open, when covered with a periosteum-lined membrane, there are other factors of bone biology that could be held responsible for and could explain such an occasional occurrence.* When a fenestra which was covered with a periosteum-lined membrane remains accidentally open, the hearing improvement, nevertheless, gradually recedes. This is due to the fact that unless the membrane employed to cover the fenestra is epitheliated on both sides, it undergoes thickening as a result of fibrosis resulting from postoperative inflammatory reaction and again impedes the mobility of the perilymph for airborne sound. The problem that should concern us is the fact that in 150 cases the epitheliated inner surface of Shrapnell's membrane was employed to cover a newly created fenestra in the external semicircular canal and the fenestra remained

permanently open in 126 of the cases thus treated. Since neither I nor anyone else has ever before succeeded in keeping the new fenestra repeatedly and permanently open following the use of various other types of membrane for covering the fenestra, and since such a large percentage of fenestrae did remain permanently open in this case, I felt that this could not be dismissed as just another coincidence but should be subjected to an investigation of the influence, if any, which Shrapnell's membrane bears upon the prevention of osteogenesis.

Having proven experimentally that bone regeneration eventually does take place in defects made in periosteal bone with the polishing and burnishing burr when covered with a periosteum-lined inner surface of a membrane, and being fortified with the observation that I succeeded in preventing osteogenesis in a vast majority of my cases by employing the polishing burr and covering the fenestra with the epithelium-lined inner mucosal surface of Shrapnell's membrane, it is perhaps not unreasonable to have assumed that since the polishing burr only retards osteogenesis and does not prevent it, the type of membrane employed to cover a fenestra thus treated is an important factor in the ultimate prevention of osteogenesis. Perhaps a periosteum-lined inner surface of a membrane, when placed in contact with the newly created fenestra, adheres to the periosteal bony walls of the fenestra and with its rich blood supply nourishes these walls and thus stimulates and restores the power of new bone formation, which has been interfered with by the employment of the burnishing burr. Whereas, when the epitheliated inner surface of a membrane is placed facing the fenestra, it does not adhere to and, therefore, does not nourish the injured bony wall of the fenestra, and thus the power for new bone regeneration which has been retarded by the polishing and burnishing burrs is not further stimulated and restored.

In an attempt to determine whether the histologic structure of the membrane employed to cover the newly created fenestra has any bearing upon osteogenesis and to prove whether a membrane whose inner surface is epithelium-lined will help to prevent new bone regeneration, while a membrane with a periosteum-lined inner surface will encourage it, I have reoperated 16 cases where the fenestra closed as a

result of new bone regeneration, in the following manner: I elevated the tympanomeatal membrane, beginning with its posterior end, for a distance sufficient to expose the entire external semicircular canal and reopened the closed fenestra

Fig. 20 (colored plate). Newly formed bone within the periosteal layer of the bony walls of the external semicircular canal fenestra resulting from excessive postoperative inflammatory reaction of soft tissues.

Fig. 21. Microphotograph of meatal portion of tympanomeatal membrane showing hyperplasia.

within it. I then obtained, from the skin of the thigh, the thinnest possible Thiersch graft, large enough to cover the bony surface of the entire external semicircular canal, and placed it with its epithelium-lined surface facing the fenestra. By replacing and molding with paraffin mesh gauze the elevated portion of the tympanomeatal membrane to its formerly occupied position, that portion of its inner periosteum-

lined surface which faced the external semicircular canal came into direct contact with the freshly-cut bleeding surface of the Thiersch graft. Due to the rich blood supply of the tympanomeatal membrane, the Thiersch graft quickly became adherent to the inner periosteum-lined surface of the tympanomeatal membrane, thus converting the portion of the tympanomeatal membrane facing the region of the external semicircular canal fenestra into a membrane whose inner and outer surfaces were both epitheliated.

In 12 of the cases reoperated upon in this manner, the fenestra remained permanently open. The oldest case was thus treated nine months ago and the fenestra has remained open. In one case the fenestra was revised four times. Not until the Thiersch graft was employed during the fourth revision did the fenestra remain permanently open. I do not agree with Sourdille[3] that repeated fenestration results in the exhaustion of the bone regeneration power. My observations have led me to believe that the opposite is true. I have observed that with each revision of the fenestra, new bone regeneration took place much more rapidly than following the first fenestration. I believe that following the first fenestration, the normal inflammatory tissue reaction which followed postoperatively left the tissues involved much richer in blood supply, thus making the tendency to repair much stronger, rather than weaker.

Having met with such remarkable success in employing this technique for the revision of closed fenestrae, after failing in all my previous attempts to keep the fenestra open following revision by replacing the periosteum-lined inner surface of the tympanomeatal membrane over the fenestra, I operated upon seven cases of otosclerosis without making a special effort to cover the fenestra in the external semicircular canal with Shrapnell's membrane. Instead, I converted the inner periosteum-lined surface of the meatal cutaneous portion of the tympanomeatal membrane nearest and attached to Shrapnell's membrane into an epithelium-lined surface with a Thiersch graft. In five of the cases thus treated, the membrane sloughed away and the fenestra closed quickly. The fenestra remained open only in two of the cases thus treated. Evidently the application of a Thiersch graft to the thinnest portion of the tympanomeatal membrane is too great

a strain upon the delicate blood supply of this membrane and, therefore, results usually in sloughing.

The fact, however, that this method of operation is so consistently successful in revisions suggests that this technique could be successfully employed in a two-stage fenestration operation. By permitting an interval of six months to pass between the first and second stages, the tympanomeatal membrane, after having undergone postoperative inflammatory changes, is much thicker in the region facing the external semicircular canal than it was originally and its periosteum-lined inner surface is much richer in blood supply. Under these circumstances, the Thiersch graft can be employed successfully in the manner above described during this second stage.

To further prove the importance of covering the fenestra with an epitheliated surface of a membrane, animal research is now being conducted by Dr. Philip Meltzer, Dr. Spector and myself in the Otologic Research Department of the Tuft Medical School, through the benevolence of the Dazian Foundation.

IS AN AIR SPACE ESSENTIAL FOR THE IMPROVEMENT OF HEARING BY FENESTRATION?

Practical serviceable hearing cannot be permanently restored by fenestrating the external semicircular canal and covering the fenestra with a membrane unless the rest of the air conduction mechanism is also reconstructed in order to physiologically best serve the new fenestra. *In order to raise by fenestration the air conduction hearing for the conversational frequencies to the decibel level necessary for serviceable hearing, it is essential to maintain the tympanic air space hermetically sealed with the tympanic portion of the tympanomeatal membrane and a patent Eustachian tube to regulate the air pressure within it.*

The tympanic air space should be widened to include within it the fenestrated external semicircular canal. The amputation of the head and neck of the malleus is essential to permit the hermetic sealing of the widened tympanic cavity with the tympanomeatal membrane to the exclusion of the epitympanum. The short process of the incus should be main-

tained in its position within the fossa incudis. The short process of the incus normally protrudes further outward from the widened tympanic air space than the external semicircular canal fenestra which lies posterior and internal to it. *It is this relationship in the positions of the incus and external semicircular canal which prevents Shrapnell's membrane from becoming adherent and fixed to the external semicircular canal fenestra, when the tympanomeatal membrane is placed in position, thus leaving a minute air space between Shrapnell's membrane and the fenestra.* As a result of this, the continuity for free mobility and vibration between the membrana tensa and the membrana flaccida, which covers the fenestra, remains uninterrupted and both respond, as a whole instead of separately, to air-borne sound, with no interference to the transmission of vibration from the membrana tensa to the membrana flaccida, which in turn mobilizes the perilymph and endolymph through the fenestra in the external semicircular canal.

The infinitesimal give of the short process of the incus in its fossa incudis, with which the tympanic membrane is now in direct contact, helps to increase the mobility of Shrapnell's membrane. That such a mobilization of Shrapnell's membrane is really accomplished as a result of a successful reconstruction of the air conduction apparatus may be seen from the fact that sneezing or blowing of the nose produces a momentary nystagmus in the successfully operated upon cases.

Whenever, after having amputated the head and neck of the malleus, the incus was also removed, it was much easier to extend Shrapnell's membrane to cover the fenestra in the horizontal canal, but it invariably became adherent to the walls of the fenestra, thus resulting in an isolation of the fenestra from the rest of the air conduction mechanism and fixation of the entire tympanic portion of the tympanomeatal membrane. In these cases, sneezing or blowing of the nose did not result in momentary nystagmus. The hearing in these cases, though better than preoperatively, never reached the necessary level to be of practical value to the patient. The fenestra closed rapidly in such cases because the epitheliated mucosal surface of Shrapnell's membrane, by adhering to the bony walls of the fenestra, rapidly became fibrotic and stimulated new bone regeneration.

HOW CAN WE PREVENT EXCESSIVE POSTOPERATIVE INFLAMMATORY SOFT TISSUE REACTION?

Postoperative inflammatory reaction of the soft tissues is the greatest deterrent to the permanent restoration of serviceable hearing in otosclerosis. It is for this reason that every step in the technique must be performed with the least amount of tissue sacrifice and the least amount of trauma to the tissues involved. This surgery must be performed under the strictest rules of asepsis and the postoperative wound must always be treated with sterile precautions. Unless the postoperative inflammatory reaction of the tissues is held down to an absolute minimum, new bone regeneration will take place and close the external semicircular canal fenestra in spite of all the technical precautions taken to prevent osteogenesis. The most serious sequel of excessive postoperative inflammatory soft tissue reaction is serous labyrinthitis. As a result of a severe postoperative inflammation, the tympanomeatal membrane may become necrotic and slough away, thus permitting fibrous tissue to extend from the mastoid wound into the fenestra and perilymph space.

The technique which I employ results in a minimum of postoperative inflammatory reaction of soft tissues because the endaural approach is one of the essential features thereof. With the endaural approach to the surgery for otosclerosis there is less tissue sacrificed and less tissue traumatized than with the postauricular or combined endaural-postauricular approach as employed by Sourdille.[3] The reason Sourdille, in using his technique, has to operate in three stages in six-month intervals is, as he himself admits, because he has to combat infection resulting from the severe postoperative inflammation of the soft tissues following each surgical intervention.

The high degree of postoperative inflammatory reaction of soft tissues, which must inevitably follow the employment of the combined endaural-postauricular approach to otosclerosis surgery, will most always result in closure of the fenestra by new bone formation, especially when this approach is applied to a one-stage procedure. Every otologist who has been trained in, and is employing, the endaural fenestration technique will admit that the endaural approach offers an

easier and better accessibility to, and a more direct visibility of, the operative field.

Those otologists who object to the endaural approach because of their reluctance to abandon the postauricular route, to which they have become accustomed, will soon find that if they wish to obtain good permanent results with otosclerosis, they will have to abandon not only the postauricular approach but many other surgical habits which they have acquired in their career of surgery for suppurative lesions of the temporal bone. In treating disease of the temporal bone, the otologist, in order to obtain the desired result, must eradicate the disease; and then permit and even encourage the reparative processes to occur. Whereas, in surgery for the improvement of hearing in otosclerosis, we attack a normal healthy temporal bone with the normal reparative powers at their best and must do everything possible to discourage and prevent this natural tendency-to-repair from occurring. That is where the difference lies between surgery for suppurative lesions of the temporal bone and surgery for otosclerosis.

When the endaural fenestration is performed correctly and meticulously. the newly created fenestra in the external semicircular canal remains permanently open. This technique prevents osteogenesis because it is based upon surgical principles which take cognizance of the biologic and physiologic factors involved in the surgery for otosclerosis.

LEMPERT FENESTRATION VS. HEARING AID.

1. Fenestration of the external semicircular canal restores physiological hearing function to the deafened. A hearing aid amplifies the spoken voice without improving the hearing function.

2. There is sufficient evidence today that the progression of the hearing loss which accompanies otosclerosis, is retarded and perhaps even checked, as a result of the fenestration of the external semicircular canal. The use of a hearing aid permits the loss of hearing to continue to progress unabated until it reaches the stage where the hearing aid ceases to be of any value, and it is then too late to institute surgical therapy.

3. Tinnitus, which is the most distressing symptom accompanying deafness from otosclerosis, is eliminated following a successful fenestration operation. Whereas, when the hearing aid is employed, the tinnitus remains unchanged.

4. Fenestration restores the hearing intelligibility for group conversation. The hearing aid is confusing in group conversation.

5. Following fenestration, the patient can hear conversation or any other sound coming from any direction, even when not directed at him. With the hearing aid he can only hear person-to-person conversation when directed towards the receiver of the hearing aid.

6. Following fenestration, conversation is heard as it is normally spoken, while conversation heard with the hearing aid is distorted in tone.

7. The restoration of physiological hearing, as a result of fenestration, changes the entire mental status of the patient, whereas the wearing of a hearing aid depresses the mental state of the patient still further.

8. The hearing aid cannot be employed for direct telephone conversation.

9. The social and economic advantages obtained as a result of the successful fenestration operation are limitless. The hearing aid is a deterrent to social and economic rehabilitation.

10. The use of hearing aids cannot be compared to the use of visual aids because we are expected to hear conversation or any other sound directed towards us or anyone else, in and from any direction, even in our sleep. Such hearing protects us and guides us in life's hazards and is also necessary in our daily economic life, whereas we are only expected to see when we are directly making a special effort to look in the direction of the object we are trying to visualize. As a matter of fact, even ophthalmologists do not advise the use of visual aids for visual defects requiring surgical measures.

CONCLUSIONS.

1. Surgery for the improvement of hearing in otosclerosis is no longer in the experimental stage.

2. There is no surgical risk to life involved in the fenestration of the external semicircular canal for the restoration of practical physiological hearing in otosclerosis when this surgical procedure is performed under the strictest rules of asepsis.

3. The amount and nature of the discomfort a patient is subjected to as a result of this surgery compares favorably with any other elective major surgical procedure and is disproportionately small when compared to the physical, mental, social and economic benefits derived from such surgery when it results in the restoration of practical hearing.

4. As a result of the Lempert fenestration, practical physiological hearing can be permanently restored in about 80 per cent of properly chosen cases of otosclerosis. The success of this work, of course, will always vary with the skill and patience of the operator.

5. This surgery should be judged by the great percentage of successful permanent results obtained therewith and not by the occasional failures which must of necessity, because of the human variable, accompany every surgical procedure.

6. This operation should not be regarded as just another operative technique added to the list of operative procedures on the temporal bone. This operation blasts a trail for a new and different type of surgery on the temporal bone. It is based upon different surgical principles than any surgery heretofore employed for the relief of suppurative lesions in the temporal bone. The best results from this surgical procedure will be obtained by otologists who, in addition to an extensive experience in all surgery upon the temporal bone, are possessed of thorough knowledge and understanding of the surgical principles of plastic reconstructive surgery.

7. In fairness to the already successful development of the surgery for otosclerosis and for the protection of its still brighter future, no otologist, no matter how skillful a surgeon he may be, should attempt this particular operation without having received special training in this type of surgery under supervision and guidance.

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PLASTIC SURGERY OF THE NOSE.*†

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I take it for granted that you all know the basic principles of correcting the nasal contour, especially the simple types, and will, therefore, not go into great detail on this phase so as to have time to spend on phases that present special problems.

The building of an entire new nose or part of a nose by means of forehead flaps, or flaps from the arm or shifting flaps from nearby structures will not be discussed in this paper. The noses that will be discussed are those that have all their component elements. The component elements are either in excess or lessened or displaced. In reference to indications for this kind of corrective surgery, it is only necessary to state that they are both psychologic and physiologic, of which the psychologic is the more important. Many nasal disfigurements appear small to the examiner but to the patient are very large and important, and mar their lives psychologically. With our present knowledge and experience in this field, these can now easily and satisfactorily be corrected in the vast majority of cases.

Our ultimate aim today is to get as nearly a perfect result as possible. People who have undergone a nasal plastic become markedly nose conscious and look for all possible flaws. It is important to keep these patients under observation for about a year before discharging them. Postoperatively, their noses undergo changes for a long time. The subcutaneous contraction and resultant molding of the bony and cartilaginous framework can so markedly affect the organ that an almost perfect result in the operating room will show distinct flaws several months later. It is a good rule, therefore, to tell all patients two things: *1.* That one or more additional procedures may be necessary; *2.* that they will have to come for observation for a long time.

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*Read by invitation at the meeting of the Section on Otolaryngology, College of Physicians, Philadelphia, Dec. 20, 1939.

†From Department of Laryngology, Graduate School of Medicine, University of Pennsylvania.

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The dislike for this work formerly manifested by the medical profession in general has now been replaced by a great deal of interest in the subject. This is very helpful and is producing freer discussion and better results. It is my belief that nasal plastic surgery is best performed by those who have had a good rhinologic training.

In studying noses for corrective purposes, inspection must be carefully followed by palpation. Palpation is a great aid in the study of the pathologic anatomy; the underlying cause of the nasal deformity. *Example:* Given a nose with a simple anteroposterior septal angulation to the left and an external bulging to the left, palpation will show the left side to be hard and resistant, while the right side is soft and not resistant. A nasal tip with a displacement of the septal cartilage from the vomer and columella will feel soft and be easily pressed backward.

The methods of correction have undergone drastic changes in the past decades. A favorable method for removing a hump was to make an external incision along the side of the nose, separate the periosteum and perichondrium, with its skin covering; with a saw, chisel, knife or other instruments remove the hump and replace the skin covering. If the removed hump was not too large, the skin adjusted itself and the result was some improvement. Another method of removing the hump was to make a columellar incision and through it lift up the skin over the hump and sides of the nose, and by means of a powerful pair of scissors or chisels, the hump was removed. The fact that the nasal bridge was left too broad did not at first bother the surgeon, but it did the patient, and he or she were usually dissatisfied. A method had to be found for narrowing the bridge. The method of Marshall, of Philadelphia, came into vogue. This method for narrowing the bridge is still used by many men today. It consists of making a small nick in the skin somewhere along the cantholabial line down to the periosteum, and by means of a sharp chisel and a few light mallet taps, the bone is broken through at this spot. The chisel is then pushed upward through the same nick, and another cut in the bone is made. This is continued up and down the cantholabial line until the entire nasal process of the superior maxilla is fractured through. This procedure is repeated on the opposite

side, and by firm pressure both fractured pieces are pushed toward each other, thus narrowing the bridge.

Finally, the intranasal method of lifting the nasal skin and attacking the bony and cartilaginous structures was accepted by almost all of us. Here, an intranasal incision between the upper and lower lateral cartilages is made on each side (the so-called intercartilaginous incision), the skin structure is elevated, and correction of the bony and cartilaginous nasal framework continued.

The procedures thus far still left much to be desired. The noses were too long, so a triangular piece of septal cartilage with its mucoperichondrium on each side was removed at its lowest border and the columella then resewn to the septum. The nose was thus made shorter, but still several things were wrong. The movable or cartilaginous part of the nose was too broad, and the shortness of the nose that showed up so well on the operating table did not stay short. Why? Because the upper lateral cartilages were not touched. The lower ends of the upper lateral cartilage reach almost to the anterior superior angle of the septum. The lower part of the septum was removed by the triangular section but the prong-like projections of the upper lateral still remained and kept pushing the tip downward, so a late postoperative droop was noted. It is to be remembered also that these upper laterals help greatly in producing the width of the cartilaginous nose. This conception of the function of the upper laterals once having been recognized, the removal of wedges of each upper lateral was performed whenever a nose was shortened. As a result the nose remained short and, also, its width was lessened. We now, therefore, have a technique that will correct the long nose, remove the hump and narrow the bony and cartilaginous section. This, however, was still insufficient because we should always bear in mind that those who undergo an operation for correction of the nasal contour become superconscious of this organ and want as near perfection as they can get, and will go to extremes to get it. Personally, I am usually in accord with this view of the patient and will do several procedures to get an ultimate result that I think suitable for the individual.

The nasal tip has not as yet been touched, and if a nose is shortened and narrowed, the nasal tip usually has to be nar-

rowed also. This is done by removing small sections of the lower lateral cartilages, care being taken not to remove too much. We work here in millimetres, not inches. Many a good result is marred by a poor nasal tip.

For the development of this entire intranasal technique, one figure stands out pre-eminently; that of Jacques Joseph, of Berlin, who first described the complete intranasal technique, and who is correctly called the father of intranasal plastic surgery.

Fear of infection, constantly reiterated by most exponents of extranasal incisions, was soon put aside as almost groundless, and now we rarely see extranasal incisions used.

The saddle nose was corrected years ago, also, by an extranasal incision (either across the columella or at the inner eyebrow or at the glabellar nasal groove), and the insertion of a transplant of bone or cartilage, or bone and cartilage, and even ivory. This has given way to an intranasal incision and the placing of these transplants through it, with very good results and no external scar. The transplant material chosen by most surgeons is cartilage. Most men avoid all foreign bodies. The use of paraffin, in great vogue two decades ago, is mentioned only to be avoided as dangerous, and gives results that are poor and not lasting.

The technique of correcting the saddle nose has been improved greatly in the past few years. Careful examination of these cases shows pathologic conditions in addition to the depression. The bony bridge and the upper lateral cartilages may be unduly wide. The nasal tip is occasionally too short anteroposteriorly. The tip structure may be soft. A transplant inserted raises the bridge and makes the nose look better, true, but this often does not meet the patient's requirements.

Some saddle noses are very difficult to correct, because, in addition to the depression of the nasal dorsum, there is a sinking-in at the columellar philtral junction. These faces are commonly called dish faces, but it is to be remembered that a columella-philtral depression may be present without a saddle nose. In the slide to be shown, a good result was obtained by placing a piece of ivory in front of the anterior nasal spine, in addition to the dorsal implantation (this case

was performed 12 years ago). Dr. Sheehan corrects these dish faces by placing pieces of cartilage around the lower nasal pyriform edge. I corrected another case by lifting the septal cartilage from the vomer, and placing between it and the vomer a strip of cartilage, which was also made to protrude slightly at its lower edge. Many low saddle noses are seen following a submucous resection, due mainly to faulty technique or postoperative infection. Such a case, if discovered while doing the submucous, is easily corrected by the implantation of a piece of the removed septal cartilage. If discovered at a later date, a piece of cartilage taken from the patient's auricle or saved from another patient will give a good result.

Small depressions are often noted when the operation is about finished. These can be corrected and the nasal contour evened up by implantation of one or more pieces of cartilage that have been removed during the operation, either from the septum or the lateral cartilages, and which have been carefully cleaned of all perichondrium. This produces a good result in the operating room, but months afterwards the nasal contour undergoes changes, and the small cartilaginous implants may produce a small local elevation, with a nearby depression. The reason for this is that it takes a long time for the nasal contour to assume its permanent shape. The nose that looked perfect on the table, a year later may show a flaw. These changes may occur because of some absorption of the cartilage and its replacement by fibrous tissue, in addition to the gradual skin contractions. This continuous gradual change due to intranasal and skin postoperative adjustment is especially to be kept in mind in those cases that have been operated upon for bent cartilages, where a superimposition operation of some kind was performed. Here, frequent adjustments by intranasal manipulations or extranasal pressure and splints must frequently be resorted to. The pull in different directions of intranasal scar tissue is unpredictable and must be frequently regulated during the course of treatment.

A. Correction of a Broad Saddle Nose:

In a broad saddle nose, the following technique gives good results: *1.* Via an intercartilaginous incision, the skin over the nasal dorsum is elevated. *2.* The upper laterals are cut

on each side from their connections to the septum. *3.* An appropriately-sized section of both nasal bones from their pyriform edge to the nasoglabellar junction in the center is separated from the nasal bones and frontal bone, by means of a proper-sized gouge, and then left in its position. *4.* The nasal processes are sawed, fractured and pushed medialward so that their medial edges go under the free bony piece and lifts this bony piece forward. *5.* The upper laterals are sewed to each other over the septal cartilage. If the nasal dorsum is not high enough, a transplant is inserted. This is

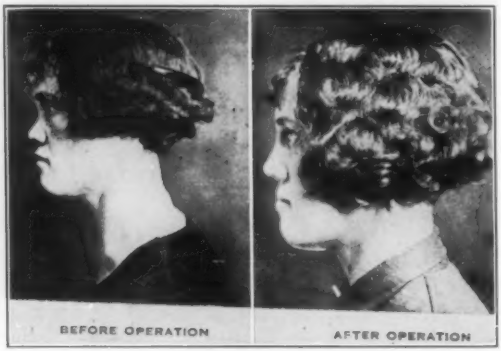

Fig. 1.

usually of auricular cartilage folded on itself or, if a still larger piece is needed, a piece of rib cartilage can be used. If necessary, the lower laterals may be corrected, both to increase the anteroposterior diameter or narrow the tip.

B. Correction of Abnormality of the Nasoglabellar Angle:

The normal nasoglabellar angle is about 145°. A much shallower angle is disfiguring and needs correction. The use of a special rasp, like the Aufricht, deepens this angle a little but usually that is insufficient. The following technique is advised: *1.* A narrow strip of bone consisting of an equal part of each nasal bone from the pyriform margin to the glabella is removed. This can be accomplished by upward chiseling with a narrow gouge, then grasping the bony strip with a pair of ethmoid forceps, and by rocking and pulling it can be removed. If it does not come easily, a few taps of

a small, sharp chisel at the glabella, internally or externally, will free it. *2.* The nasal processes are then sawed or chiseled and pushed towards each other. The result will be a deeper nasoglabellar angle and a narrower bony nose. The removal of the central bony strip can also be accomplished by simple straight chisels working upward on each side at an equal distance from the center line. Then the same chisel is used on the flat and the perpendicular plate, and part of the septal cartilage is cut away from its junction to the intranasal suture line (posteriorly). *3.* The strip of nasal bone is then

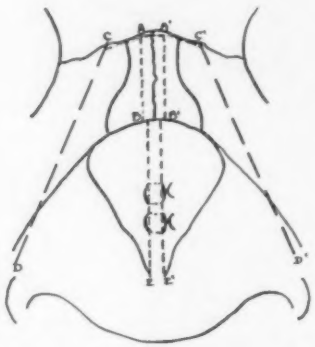

Fig. 2. Marked saddle nose. Also for nasoglabellar angle and flattened nose. Incision (ab), (a), (b) and (a-a) set free united pieces of nasal bones which are left in place. (cd), (c) (d) incisions on each nasal process which are fractured and pushed towards each other so that the medial edges are pushed under the free central strip (ab-a) (b) (-a) (a). (be) and (b) (e) are separations of each upper lateral from septal cartilage and sewed to each other. An additional implant is needed (text).

removed, as mentioned before. *4.* If a hump is part of the condition to be corrected, the hump is removed first. Immobilization by proper postoperative dressings must be kept up for about one week.

C. Correction of Bulging of Upper Lateral Cartilage on One Side:

This is a frequent nasal deformity. Here, the nose is well formed, except that one side has a bulge, which on examination is found to be due to a bad septal angulation pushing that side lateralward. The ordinary submucous; that is, removal of a large piece of septal cartilage, including the

angle, and leaving a strip low down and another strip anteriorly, the so-called support strips, will result only in some improvement to breathing and little, if any, correction of the external deformity. The following technique is suggested: *1.* An incision is made anteroposteriorly, slightly in front of

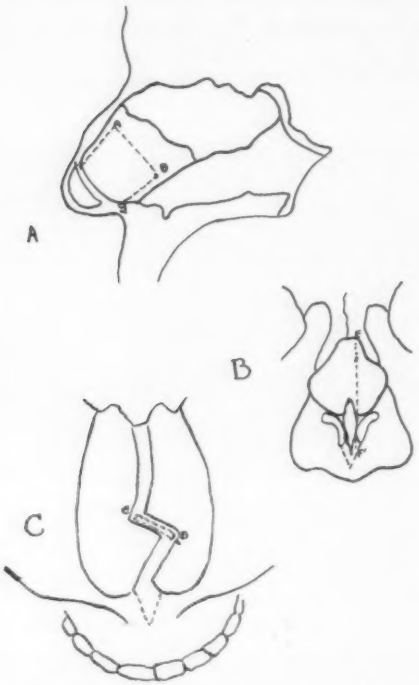

Fig. 3. Lower nasal deviation to left, bulging of left upper lateral. A—(a-b) incision of mucosa through cartilage to opposite perichondrium. (a-h-b-g) incision through cartilage extended forward, producing mucocartilaginous flap. B—(F-F') left upper lateral cut from septum. C—(c-d) section of cartilage removed from (a-b) (a-h) and (b-g).

the septal angle, all the way down from the nasal dorsum to the vomer, and the septal cartilage is removed submucously above this. It is removed almost entirely to and including part of the perpendicular plate, to the vomer, and to almost its union with the upper laterals. A saddle nose will not result, as the upper laterals will prevent this. *2.* A second

incision is made through the mucous membrane and cartilage, to but not through the opposite mucous membrane, along the nasal floor from the posterior end of the first incision to the septal membrane. *3.* A third similar incision is made parallel to the anterior septal border, far forward,

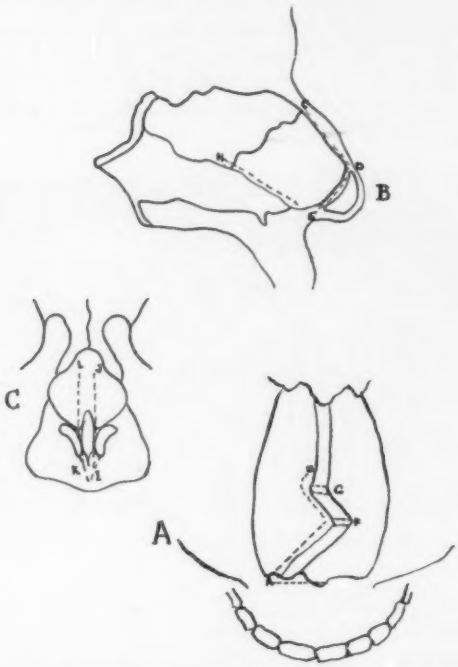

Fig. 4. Displaced septal cartilage. A—(a-b) mucosa and perichondrium elevated. (f-g) strips of cartilage removed at angulations. B—(c) (d), (e) elevation of skin over lower nasal dorsum, and separation of columella from septal cartilage. (e), (h) septal cartilage lifted off vomer. C—(i), (j) and (k), (l) incision separating each upper lateral from septal cartilage (text).

through the mucous membrane and cartilage, from the first incision to the septal membrane. This quadrangular piece of septal cartilage can now easily be pushed medialward and centralized. We now have a straight septum, but the bulge of the external nose on that side is still present and will not disappear of its own accord, even though the original cause of the nasal deformity has been removed. The upper lateral

cartilage, which is bulging, must be separated from its attachment to the septum. This is done by separating this upper lateral with its lining mucosa from the skin; then, with a scissors cut its medial (septal) attachment. Slight external pressure for several days, in addition to the usual attention to this modified submucous, will give a good nasal configuration and good breathing (sometimes crisscrossing the cartilage is also necessary). If the vomer protrudes, a small piece of it can be chiseled under and pushed over with the quadrangular piece of septal cartilage. If it does not come over very easily, a little separation of the perichondrium on the

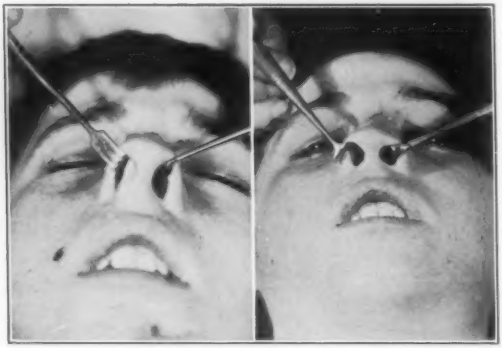

Fig. 5. Dislocated cartilage, before and after correction.

opposite side, anteriorly and posteriorly, will help. This quadrangular flap, which consists of a piece of cartilage and attached mucosa on both sides, must move centrally with ease and remain where it is placed. On the third day postoperatively, it will be well to insert a perforated splint into the previously obstructed nostril, and the patient should wear this for about one week, in addition to daily pressure treatments to keep the bulge reduced.

D. Correction of Flattened Lower Nose:

There are noses where the lower or cartilaginous part presents an anterior flatness. The following gives a fair result. The skin over the cartilaginous and bony nose is elevated and the columella is cut from the lower border of the septal

cartilage. This is accomplished via the usual intercartilaginous incision. The upper laterals are then cut from their attachment to the septum. The medial border of the upper laterals are brought together and sewed to each other with fine mattress sutures over the septum. This procedure is usually combined with other steps in nasal plastic surgery.

E. Correction of Dislocated Septal Cartilage:

The correction of the dislocated septal cartilage is, in brief, as follows: An intercartilaginous incision is made and the

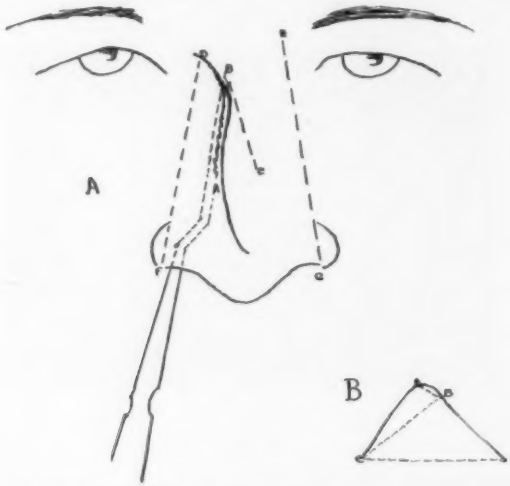

Fig. 6. Twisted bony nose to right. A—(a-b) saw cut at top of bony deviation; saw points posteromedially. (b-c) saw cut on opposite side; saw points anteromedially. (d-f-e-g) saw cuts on each nasal process. B—(a-b) deviated hum removed at an angle—wall (ac) will be removed to position (bc).

skin over the lower nose is elevated. The septum is cut from the columella. The upper lateral cartilages are cut from their septal attachments. The mucoperichondrium is separated on the concave side of the dislocated septal cartilage all the way up to the perpendicular plate. Two slivers of cartilage in an anteroposterior direction are removed, one at the first or lower angle, and another at the second angle higher up. The septal cartilage is separated from the vomer. The three pieces of septal cartilage now present are placed in the center. The

columella is partly divided on its upper surface, and the septocolumella gap is closed by two or three silk sutures. In adults it is often good to remove the entire septal cartilage above the first angulation, submucously, leaving only the lower section, as described previously, for angulated septal cartilage with an external bulge.

F. Correction of Deflected Bony Noses:

The deviated bony nose is corrected by most operators as follows: A triangular strip of bone of the wider side, base at the pyriform margin, is removed, in addition to a strip at

Fig. 7. Before and after operation.

the bony dorsum, then the nasal processes are fractured and brought towards each other. Several years ago at a meeting of the Society of Plastic and Reconstructive Surgery, I presented the following procedure: The usual intercartilaginous incision, skin elevation and separation of columella from the septal cartilage is performed. The bony hump present in these cases is next removed at *an angle* so that the bony sides of the nose that are left are about equal in width. The nasal processes are then fractured and pushed towards each other, giving a fairly straight centralized bony nasal dorsum. The important point here is that the saw is first placed on the convex side, at or near the top of the ridge, and instead of sawing straight across the nasal bridge, the sawing proceeds at an angle posterolaterally, so that if continued all the way

it will come out on the concave side more posteriorly. There are various modifications and explanations to this procedure, and it requires a separate paper, which will shortly be published.

G. Correction of Abnormal Wide Nasal Floor:

Most operators correct a wide nasal base with wide nostrils by the Kolle method. Here, a diamond-shaped piece is removed from the floor of each nostril and upper lip, with a maximum width at the margin of the nostril. My objection to this procedure is, first, that two scars are left on the upper lip; second, a narrowing of the nostril has occurred in one

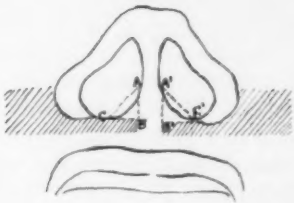

Fig. 8. Triangles (abc—a b c) are removed from nasal floor down to the bone. Shaded areas denote undermining over each maxilla (text).

of my cases, causing my patient some breathing difficulty; third, the Kolle technique may pull the lower end of the alae medially, but not their upper ends; fourth, the deformity may redevelop. The following procedure is suggested: The floor of each nasal vestibule is elevated laterally off the bone from the anterior nasal spine and vomer to about one inch outside of each ala (under the cheek), then a triangular strip of this is removed on each side of the central line. The medial edge of each flap is then stitched to the septal membrane on its side. If protrusion of the upper lip is needed, the medial edges are brought together in front of the anterior nasal spine. The alae are thus brought nearer to each other. A strip of adhesive plaster is placed from one alae to the other. This will lessen the tension of the central sutures. The adhesive strip or strips may be left on for five days.

The time when to operate is important. It seems best to wait till the patient is past 18 years and full body growth has occurred (this can be corroborated by X-ray studies of

the joints). Sometimes, however, it may be necessary to do a correction earlier. Dislocated septal cartilages can be corrected very early. I've done one in a child age 4 years, with a definite and continuous improvement. An acutely bent lower septal cartilage may cause both nasal obstruction and an external nasal bulge on one side. Here, waiting for full growth before correction to me, at least, seems a fallacy. Here, a modified submucous, as described, wherein incisions or, at most, small slivers of cartilage are removed at the angles gives a good result and interferes little, if any, with growth. Nasal fractures should be corrected as soon as possible. If, after a nasal injury, attempts at replacement of the nasal structures by intra- and extranasal manipulation are unsuccessful, it is then best to wait two or three months, when the structures have adjusted themselves to their new position, and then operate. But by all means correct nasal fractures as early as possible. M. C. was seen by me four weeks after an automobile accident. The bony nose was pushed to the right and the dorsum was flat. Under local anesthesia, intra- and extranasal manipulation procedures replaced the bony nose centrally and also raised the nasal dorsum. This, in my experience, was the longest post-traumatic case wherein simple manipulations accomplished the desired result.

Whether to perform a submucous resection before or after a nasal plastic is not definitely agreed upon by men in the field. The majority still do the submucous first, wait one or two months and then do the plastic. I do not follow this procedure. In most cases a mild septal correction is performed at the same time as the plastic. If much has to be done, it seems best to me to first do the plastic, wait several months and then do the submucous.

Miss A had a nasal plastic performed one month after the submucous. She was observed for about two months and seemed satisfied. Two years later, examination showed flatness at the tip. Intranasal scar contraction was too strong for the weakened septal cartilage. In exceptional cases wherein a high deflection of the perpendicular plate will prevent narrowing of the nasal processes during the plastic, it may be best to perform the submucous before the plastic. Even here the perpendicular plate can be corrected at the time of the operation and a small amount of packing inserted

to keep the flaps together. In a deviated nose, that is, where the entire nose is shifted to one side, the perpendicular plate can in the main be removed while doing the plastic operation and the deviated septal cartilage centralized.

All our nasal plastics are performed under local anesthesia, mainly infiltration in type. In addition, the infraorbital nerve at its exit and the external nasal nerve at its exit are injected via a needle puncture, about one-eighth inch lateral to each ala, and another needle puncture at the central part of each pyriform margin. A few drops of novocain are injected on each side of the anterior nasal spine. One hour before the operation, the adult patient is given 3 gr. of nembutal, and just before being wheeled into the operating room, a hypodermic injection of morphine gr. $\frac{1}{4}$ and atropine sulphate gr. 1/100.

Complications due to poor technique or poor judgment are frequent. Complications due to infection are infrequent and rarely serious. Here, a knowledge of rhinologic treatment is very important and again demonstrates the previous statement made that those undertaking nasal plastic surgery should have a good training in rhinologic treatment, both medical and surgical.

In conclusion, permit me to say that in order to obtain as perfect a nose as possible, many problems have as yet not been worked out and publicized, especially in areas like the nasoglabellar angle, nasophiltral angle, and nasoalar curve, nasal tip, etc. The frequent need of secondary corrections probably demonstrates the necessity for further improvement in technique. Yet, progress has been made and usually the results are gratifying.

2009 Pine Street.

CELLULITIS OF THE FACE WITH OSTEOMYELITIS OF THE FRONTAL BONE AS A COMPLICATION.*

DR. O. W. THOENY, Phoenix.

This patient is B. A., age 6 years at the time his illness began. He had been in good health all his life and had had a tonsillectomy a year previously as his only illness.

A day or two before admission he had developed a sore spot on his nose and this extended rather rapidly, so that when seen at noon on May 5, 1938, at the request of his physician, W. P. Sherrill, he had a well developed cellulitis, originally on the nose but extending by this time up over the forehead and showing marked edema of both lids. There was much prostration and the patient was moved into St. Joseph's Hospital immediately. His temperature at that time was 103° and rapidly rose to 104°.

His blood count showed 90 per cent hemoglobin and 24,000 white cells, of which 81 per cent were neutrophiles. The urine showed 2+ acetone and diacetic acid.

Hot boric compresses were applied over the area and he was given infrared light treatments. Sulfanilamide was started at once, gr. 10 every four hours, and fluids were forced.

Beginning the next day and at frequent intervals thereafter he was given transfusions, receiving in all 1,000 cc. of blood in five transfusions. The white count advanced to 35,700, while, in spite of the transfusions, the hemoglobin dropped to 70 per cent and the red cells to 3,200,000.

A blood culture showed a streptococcus present; within six days the blood culture was negative.

On May 8, patient was given X-ray therapy, receiving 180 r. at 100 kvp. and 1 mm. Al. filter, over the area of involvement, the eyes being protected. By May 11 there had been considerable improvement, as shown by localization

*Presented at the Eye, Ear, Nose and Throat Meeting of the Regular Staff at St. Joseph's Hospital on April 8, 1940.

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of pus, which was drained. A third X-ray treatment was given on May 19, by which time there had been marked improvement; this radiation was at 125 kvp. 2 mm. Al. filter and w 200 r.

His condition showed steady improvement and on May 20 he had a normal temperature, which he maintained thereafter. He was discharged from the hospital on May 23.

His condition then remained quite good, but about five weeks later a swelling appeared over the left frontal area, and on July 10 he was again admitted to the hospital, with a diagnosis of osteomyelitis of the left frontal bone.

On July 12 a resection of the osteomyelitic areas of the frontal bone was done, six areas being found at the time of operation. At this time he was again given several transfusions, a total of 500 cc.

His temperature during his stay in the hospital this time was never high, being normal most of the time except for the first week, when he ran about 1° of temperature.

The operative wound healed slowly, but another abscess developed over the right side of the nose at the angle of the orbit. The patient was reoperated and the contents of the abscess evacuated but in this area no areas of bone necrosis were uncovered.

He was discharged from the hospital on Aug. 13 in good condition, and his blood picture at that time showed 90 per cent hemoglobin and 4,800,000 red cells.

Drainage from the wounds persisted for a long time. On several occasions, after they had entirely healed, another abscess would form, necessitating drainage.

A number of X-ray pictures were made serially and because of the continued improvement only conservative drainage was employed, although by the extrusion of small sequestrum at infrequent intervals, necrosis of bone was still evidently continuing.

For the last year this patient has shown continued improvement. He has had several minor illnesses, at one time having an acute otitis media, at which time he ran considerable tem-

perature and was acutely ill, but there was no extension of his bone infection.

Considering the absence of symptoms for such a prolonged time and the statement by Dr. W. W. Watkins that at the present time his X-ray findings show no deviation from normal, we can consider that this child is cured.

This case as presented is an example of one of the dangerous complications of infection about the face. The patient never had any evidence of sinus disease, had no trauma, had not been swimming and, in addition, was in vigorous health. Behrens,[1] in his comprehensive review of the subject, states that dental infections and other distant foci are rarely causes of osteomyelitis.

The infection began as a fulminating streptococcus but staphylococci were found early and remained as the offending organism. The pathway of infection seems obviously to have been along the venous channels, and the small isolated areas are explained by retrograde thrombosis as demonstrated by Furstenberg[2, 3] and emphasized by Skillern,[4] who states that the diploic venous system is exposed to any infection about the orbit, particularly through the frontal diploic vein.

In the treatment of this case the conservative method was chosen; first, because the infecting organism was considered low grade, since the general condition of the patient could be maintained at a high level; and, second, because the areas of destruction did not tend to spread or coalesce. Mosher and Judd[5] have shown that infection is present seven to 10 days before demonstrated by the Roentgen ray, so that repeated films gave us a chart of the rapidity of extension. Consequently, after the individual areas had been removed to the margin of healthy tissue, it was found that no further extension occurred. The course suggested by Behrens of fitting the treatment to the type of case would be applicable to this case. The use of Roentgen ray therapy in this case would appear to have been of much benefit.

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ACUTE LARYNGOTRACHEOBRONCHITIS WITH REPORTS OF 14 CASES WHICH CAME TO TRACHEOTOMY.

DR. GEORGE O. CUMMINGS, Portland, Me.

Acute laryngotracheobronchitis is a relatively uncommon condition. It most frequently occurs in small children and is sudden in its onset. Often following a mild upper respiratory infection, increasing laryngeal dyspnea develops which may demand tracheotomy. The initial temperature is usually high. As the disease develops, it tends to progress downward from the larynx and trachea to the larger bronchi. The secretions may be thick and viscid and tend to form crusts which, unless removed, occlude the lower airways, causing death from asphyxia. Practically all of the common bacteria have been recovered in this disease; however, the hemolytic streptococcus is most frequently found and may be associated with the staphylococcus aureus. Crusting over superficial ulcerations is frequent when the latter organism is present. It is almost impetiginous in character.

Richards[1] states that at autopsy, "The most striking change was the diffuse cellular infiltration and destruction of the mucosa of the walls of the larynx, trachea and bronchi. The histological picture is comparable to that of a cellulitis of the skin and is characteristic of the streptococcus. A second type of lesion was seen in the destruction of the mucosa and the masking of the mucosal surfaces by fibrin and purulent exudate. In those cases where a superimposed infection with the staphylococcus occurred, a more marked destruction of the mucosa and wall of the trachea could be noted as a manifestation of the necrotizing characteristics of the staphylococcus." We have noted both types of lesion at autopsy in our cases.

Laryngeal dyspnea due to this disease may be confused with croup, laryngeal diphtheria, edematous septic laryngitis and foreign bodies in the tracheobronchial tree. Spasmodic croup occurs in small children and frequently accompanies

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upper respiratory infections; the attacks, which steam inhalations relieve, usually last for short periods. Laryngeal diphtheria is generally associated with diphtheritic membrane in the pharynx and is the first diagnosis to come in mind in acute laryngeal dyspnea in small children; on this account, diphtheria antitoxin is frequently administered to children with laryngeal dyspnea, not only as it is a specific for diphtheria, but from 12 to 24 hours are necessary for diagnosis by culture. The dyspnea in laryngeal or laryngotracheal diphtheria is usually more urgent, the patients more cyanotic and toxic and the relief by tracheotomy more prompt as there is not the secretion and edema in the lower airways that there is in acute laryngotracheobronchitis. Edematous septic laryngitis is primarily a laryngeal disease, does not tend to propagate downward and usually occurs in adults. Foreign bodies in the larynx or tracheobronchial tree seldom simulate the symptoms of acute laryngotracheobronchitis but such invaders may precipitate this condition; as a rule, however, the associated laryngotracheobronchitis clears up a few days after their removal.

The bronchoscopic appearance of the mucosa of the tracheobronchial tree differs in acute laryngotracheobronchitis, diphtheria, edematous septic laryngitis, and in those cases in which foreign bodies are present. In acute laryngotracheobronchitis, due to the swelling of the mucosa, there is a loss of the ringed appearance of the airways, and in certain cases there are ulcerations and crusting. The secretions tend to be scanty and viscid. The tendency to swelling of the conus elasticus below the vocal cords, the smaller airways, the lack of immunity to respiratory infection in children may play a part in the characteristic findings in this disease. In diphtheria there is the typical production of membrane. The underlying mucosa is red, moist and velvety. The rings of the airways can be visualized as there is little swelling of the mucosa, since the infection is superficial. In acute edematous septic laryngitis of adults, the disease is primarily laryngeal. The conus elasticus shows little tendency to swelling. The airways are larger, the immunity to respiratory infections greater and the involvement of the tracheobronchial tree negligible. In laryngotracheobronchitis following foreign bodies in the tracheobronchial tree, the edema is primarily in the subglottic tissues of the conus elasticus; the

mucosa of the airways below is swollen and moist, but the rings are not obliterated. The secretion may be profuse but not viscid nor crusting.

The treatment of this disease, depending on its severity, calls for bed rest, highly humidified air by steam kettle, sulfanilamide, adequate fluid intake by mouth, clysis or infusions, frequent small blood transfusions, tracheotomy with its attending nursing care before the patient is exhausted, the use at times of normal saline, dropped from 20- to 30-minute intervals into the tracheotomy tube as an attempt to prevent drying of secretions, the use of staphylococcic bacteriophage similarly instilled in cases in which the staphylococcus is found, and bronchoscopic removal of secretions and crusts when blocking of the lower airways is evidenced by dyspnea, cyanosis or evidences of atelectasis found on examination of the chest.

A kettle steaming by a bed in an open ward is useless unless the head of the bed is inclosed by screens covered over and above and partly in front by sheets, and the steam is led into the enclosure by a hose. Preferably, the patient should be in a small room, whose doors and windows are kept closed, in which a kettle is steaming.

The picture of a patient in one of the various stages of cyanosis, with the accessory muscles of inspiration in play with the indrawing of the supraclavicular and suprasternal notches and epigastrium is well known. This struggle to obtain sufficient air is fatiguing, and the younger the child the greater should be our haste to relieve the dyspnea, as small children soon become too exhausted to respond to the benefits of tracheotomy. On the whole, tracheotomy should be preferred to intubation, except possibly in those institutions in which some physician is always at hand who is skilled in this procedure.

An adequate tracheotomy set, with the necessary instruments and supplies for this operation, should always be kept sterile and available in all hospitals. Laryngeal dyspnea may be urgent and there may be no time to hunt for instruments and infrequently used tracheotomy tubes.

Tracheotomy may be performed under a local or, if expediency demands, under no anesthesia. It is easy in thin-necked

adults, and difficult in infants. While the midline incision is in a relatively bloodless area, an anomalously placed innominate vein may cross the trachea above the sternal notch. The extension of the patient's neck preparatory to this procedure pulls upward some of the thoracic trachea and in a hurried tracheotomy, dissection may be carried downward and the tops of the pleurae and mediastinum too nearly approached. This is particularly apt to happen in short, fat-necked infants. Tracheotomy can be more deliberately carried out over a bronchoscope, which gives opportunity to observe the larynx and trachea. Dyspnea, after the first few breaths following the tracheotomy, may occur from overstimulation of the respiratory centre by oxygen, and insufflation of carbon dioxide plus artificial respiration may bring relief to the patient and surgeon. Smears and cultures should be taken from the tracheal secretion at operation and daily thereafter until the bacteriology has been definitely established.

An electric or water suction apparatus, or an asepto syringe and catheter should be at hand at operation and in the patient's room until the tracheotomy tube is removed. A sterile duplicate tracheotomy tube, Trouseau dilator and headlight should be kept by the patient. It is advisable to have a sterile bronchoscope, laryngoscope, bronchoscopic forceps, aspirating tubes, light cords and battery at hand, for they may be of inestimable value if the trachea or bronchi become occluded with secretions, membranes or crusts. If such equipment is not available, much may be accomplished by suction applied via a small rubber catheter cut squarely across the end for introduction through the tracheotomy tube into the lower airways.

If the secretions have a tendency to dry or crust, a few drops of normal saline solution may be introduced into the lumen of the tracheotomy tube as often as seems necessary and reaspirated. The similar use of staphylococcic bacteriophage has been reported by Evans.[2] Opiates should not be given as they deaden the cough reflex. Atropin should not be administered as it dries the secretions.

Intubation as a procedure cannot be entirely dismissed. Tracheotomy in small infants is not without danger (see Case 9). Richards[3] mentions the occurrence of pneumothorax

after tracheotomy — the presumption being that there is leakage of air from the tracheotomy wound to the pleural cavity. I am of the opinion that intubation via direct laryngoscopy is preferable in small infants. If they do not do well, tracheotomy can be performed. Baum[4] feels that there is less tendency to drying of the secretions and crusting after intubation as the inspired air is normally moistened by the nose.

The following tabulation of cases by Richards,[3] as reported by a number of students of this disease, is of interest and illustrates the high mortality.

TABLE I.

Name	Cases	Gross Mortalities	Trache-otomies	Deaths	Tracheotomi: Mortality	Date
Gittens[5]	32	39%	20	12	80%	1936
Smith[6]	43	9.3%	12	4	33%	1936
Baum[4]	24	41%	17	9	53%	1928
Richards[3]	28	40%	23	12	50%	1937
	127		72	37	51%	

In this series of 14 cases there were seven deaths, a mortality of 50 per cent.

The hemolytic streptococcus was cultured in four cases, two of which recovered; hemolytic streptococcus and staphylococcus aureus in five cases, one of which recovered; hemolytic streptococcus and influenza bacillus in one case, which recovered; staphylococcus aureus and staphylococcus viridans in one case each that died, and no growth in one case which recovered.

The ages of the children who recovered were 15 and 18 months, 3, 5, 8 and 10 years. Of those that died, 10, 12, 18 and 22 months, 2½, 3, 11 and 13 years.

Diphtheria antitoxin was given in seven cases as a precautionary measure.

The patients who recovered were hospitalized for seven, seven, ten, 21, and 34 and 214 days. Of those that died, two passed away at the end of the first hospital day, five on the second, and one on the third.

In those who recovered, the tracheotomy tube was removed on the fourth, seventh, eighth and tenth days. One patient wore an intubation tube for 12 days and then a tracheotomy tube for eight days. In one patient the tracheotomy tube was removed at the end of 205 days after seven laryngeal dilatations.

The following case reports show the fulminating character of disease. They have been abbreviated, so that the drama connected with tracheotomy has been omitted, but do portray many of the problems arising in the treatment of this disease. Only those cases in which tracheotomies were performed are here reported, as no true estimate of the entire numbers of such cases occurring can be gathered from hospital statistics since most of the milder conditions were treated at home.

Case 1. A Thoroughly Treated Case. Sulfanilamide, Infusions, Transfusions, Recovery:

M. M. C., age 8 years, entered the Maine General Hospital Dec. 24 and was discharged Jan. 3, 1939. The night before entry she developed an acute sore throat and was seen by her family physician at 10:00 P.M. At 3:00 A.M. she developed laryngeal dyspnea and was brought to the hospital, where a tracheotomy was performed at 4:15 A.M. over a 4 mm. bronchoscope. The arytenoids and false cords were particularly edematous, and the tracheal mucosa was red and inflamed. Secretion was aspirated from the trachea. Temperature, pulse and respirations were 105°, 160 and 30. They dropped sharply the next day, and gradually returned to normal in eight days. She was given 20,000 units of diphtheria antitoxin on admission, and 30 gr. of prontylin at once, and 10 gr. every four hours for the first three days. The sulfanilamide determination in the blood was 3.5 mg. Thereafter she was given 5 gr. every four hours for two days, when it was stopped. She also received 200 cc. of citrated blood on Dec. 25 and 27. She received infusions of glucose on Dec. 25, 26 and 27. She was decannulated in eight days. Culture showed staphylococcus aureus and bacillus influenza.

Case 2. A Thoroughly Treated Case. Bronchoscopies, Sulfanilamide, Infusions. Death:

S. S., age 22 months, entered the Maine General Hospital Nov. 30 and died Dec. 3, 1939. Four days before admission she had a mild croup, which cleared up. The day before admission she was unusually well, but that night developed a rapidly increasing laryngeal dyspnea. On entry to the hospital her temperature, pulse and respirations were 103, 140 and 30. Mirror examination showed a membrane below the vocal cords, and she was given 10,000 units of diphtheria antitoxin. A tracheotomy was performed over a 4 mm. bronchoscope at 4:00 P.M. The larynx above the level of the cords was injected, and the subglottic area markedly edematous and covered with exudate. The tracheal mucosa was mildly inflamed. The administration of prontylin was begun and normal saline was dropped into the tracheotomy cannula every 20 or 30 minutes and reaspirated in an endeavor to keep the tracheal secretions

thin. Infusions of 5 per cent glucose solution were administered to prevent dehydration. The inner cannula of the tracheotomy tube was changed frequently. At 12:00 midnight and at 5:00 A.M. the entire tube was changed. Thereafter 11 bronchoscopies were performed through the tracheotomy wound, as dyspnea developed, for the removal of secretions and crusts. Finally, the smaller bronchioles began to fill, and the child died from asphyxia and toxemia. Culture showed hemolytic streptococcus and staphylococcus aureus.

Case 3. An Overwhelming Infection. Death. Autopsy:

Comment: This was a fulminating disease. He did not have the benefits of bronchoscopic removal of crusts and secretions, but it is doubtful if they would have been of value.

H. LeC., age 11 years, entered the Maine Eye and Ear Infirmary Feb. 4, and died on Feb. 5, 1934. Following an acute upper respiratory infection, he developed an increasing laryngeal dyspnea the night before admission. His temperature, pulse and respirations were 106, 150 and 38. He was markedly toxic. A tracheotomy was immediately performed and despite frequent aspiration of secretion he died of respiratory failure. At autopsy the trachea was spotted with crusts over superficial ulcerations. The smaller airways were occluded by swelling and crusts. Culture showed hemolytic streptococci and staphylococcus aureus.

Case 4. Recovery:

R. J. R., age 3 years, entered the Maine Eye and Ear Infirmary Dec. 7, and was discharged Dec. 14, 1932. Following a respiratory infection, he developed a sore throat the day before entry to the hospital, when he presented a picture of marked laryngeal dyspnea. A tracheotomy was immediately performed under local anesthesia. His temperature, pulse and respirations were 103.4, 136 and 30; in four days' time they returned to normal. He was treated symptomatically and was decannulated in seven days. Culture showed hemolytic streptococcus.

Case 5. Overwhelming Infection. Death:

B. P., age 18 months, entered the Farrington Hospital Dec. 7, and died on Dec. 9, 1937. He had had an upper respiratory infection for a week, and developed increasing laryngeal dyspnea on the night before admission. On entry to the hospital his temperature, pulse and respirations were 101, 160 and 44. He was given 10,000 units of diphtheria antitoxin and a tracheotomy was performed with little relief. His chest was said to be clear at all times. He died of an overwhelming toxemia. Culture showed hemolytic streptococci and staphylococcus aureus.

Case 6. An Overwhelming Infection. Death:

C. B., age 3 years, entered the Maine General Hospital Sept. 26 at 11:00 P.M., and died Sept. 29, 1936, at 1:00 A.M. He had had an acute sore throat for two days, followed by increasing laryngeal dyspnea. On admission he was almost moribund. An emergency tracheotomy was performed on the ward, following which he breathed more easily. His temperature, pulse and respirations were 102, 100 and 40. They climbed to 106.5, 180 and 60 before his death. He was given 20,000 units of diphtheria antitoxin shortly before admission. He was overwhelmed by toxemia. Cultures showed hemolytic streptococcus.

Case 7. A Case Developing in the Hospital While Recovering from a Mastoidectomy. Recovery:

R. W., age 5 years, entered the Maine General Hospital Oct. 25, and was discharged Dec. 23, 1932. He was admitted because of an acute otitis media. On Oct. 26 he had a myringotomy, on Oct. 28 a mastoidectomy; on Nov. 18 and 27, abscessed lymph nodes were opened in his neck; in the second week of December he had an acute upper respiratory infection; on Dec. 15 he quickly developed a laryngeal dyspnea, and a tracheotomy was performed on the ward. His temperature, pulse and respirations were 102, 130 and 30. He was given 20,000 units of diphtheria antitoxin. He did well and the tracheotomy tube was removed in four days. Culture showed hemolytic streptococcus.

Case 8. Intubation, Later Tracheotomy. Recovery:

Comment: Baum[4] states that intubation is to be preferred to tracheotomy in acute laryngotracheobronchitis as the inspired air is normally moistened, so that there is less tendency for drying and crusting.

E. C. J., age 15 months, entered the Maine General Hospital Nov. 16, and was discharged Dec. 17, 1931. Two days before admission she was croupy and her laryngeal dyspnea increased, so that she was hospitalized. Her temperature, pulse and respirations were 101, 150 and 45. She was given 10,000 units of diphtheria antitoxin and an intubation was performed. Four days later the intubation tube was removed, as her temperature, pulse and respirations had returned to normal. She did not breathe well and was again intubated. Eight days later it seemed safe to remove the tube at a direct laryngoscopy, which showed a relatively normal larynx; however, laryngeal dyspnea again developed and it was considered safer to do a tracheotomy. Eight days later the tracheotomy tube was removed. Culture showed hemolytic streptococcus and staphylococcus.

Case 9. Tracheotomy, Collapsed Lungs. Death:

Comment: This was an operative death and shows that tracheotomy in small infants is not a simple procedure, particularly when it cannot be done over a bronchoscope. This case also suggests that in small infants, intubation be tried first.

J. D., age 1 year, entered the Maine General Hospital March 6, and died on March 8, 1940. For four days she had had a croupy cough and was admitted to the hospital because of increasing dyspnea. For the first 24 hours she held her own, but seemed to be getting more fatigued in her efforts to breathe. Her temperature, pulse and respirations were 99, 120 and 30. This seemed like a mild case and tracheotomy was performed to assure recovery. A direct laryngoscopy showed subglottic edema. A 4 mm. bronchoscope could not be passed. When the tracheotomy was performed, what was at first thought to be substernal fat pushed up into the wound; however, in view of later occurrences I feel that in a short, fat-necked infant in our endeavor to keep away from the larynx our dissection was carried too low and the tops of the pleurae exposed. At first the baby breathed more easily, but the secretions became more viscid and the tracheotomy tubes had to be changed every

two or three hours. Her respirations became more rapid. She was bronchoscoped through the tracheotomy wound five times, but there did not seem to be enough crusting or secretion to explain the respiratory rate. In course of treatment she was given neoprontylin and infusions of 5 per cent glucose. Her temperature, pulse and respirations rose to 105, 160 and 50, and on March 8 she died of exhaustion and toxemia. An X-ray taken after death showed collapsed lungs. Culture showed streptococcus viridans.

Case 10. Overwhelming Infection. Death:

Comment: While tracheotomy did not greatly disturb this infant, would it have done better with an intubation at first? Bronchoscopic aspiration and removal of secretion and crusts was not done.

P. D., age 10 months, was admitted to the Maine General Hospital Feb. 22, and died Feb. 24, 1934. Two days before entry he had croup, and was admitted to the hospital because of laryngeal dyspnea and cyanosis. His temperature, pulse and respirations were 104, 160 and 40. A tracheotomy was performed and he breathed more easily. The secretions became more viscid and he died of an overwhelming infection and asphyxia. An X-ray taken after death revealed an area of atelectasis at the left base. Cultures showed hemolytic streptococci and staphylococcus aureus.

Case 11. Question of Foreign Body, Tracheotomy at Home, Measles. Recovery:

Comment: This is a case in which the question of a foreign body in the tracheobronchial tree had to be considered. It also shows that such a case can, if necessary, be successfully handled in the home.

J. C. M., age 10 years, was treated at home. His illness began May 23, and he was discharged June 26, 1926. He had left the dinner table and was in the yeard eating an apple when he developed laryngeal dyspnea. Fortunately, I was informed of this fact and took a tracheotomy tube with me. I was about to take him to the hospital for bronchoscopy for a suspected foreign body when he suddenly ceased breathing. An emergency tracheotomy was performed, artificial respiration started, and he revived. In lieu of a suction apparatus, rubber bulb syringes and rubber catheters were used. He did well for four days, when the tracheal secretions increased markedly, and had it not been for the splendid co-operation of two trained nurses and the judicious use of suction by rubber bulb syringe and catheter he would have died from asphyxia. The next morning he broke out with measles. His original laryngeal dyspnea probably coincided with the invasion of the disease, and the second incident with its efflorescence. Cultures ruled out diphtheria.

Case 12. Question of Foreign Body or Allergy, Resemblance to the Edematous Septic Laryngitis of Adults. Death. Autopsy:

Comment: The course of the disease, the appearance of the larynx, the extensive lymphadenitis before death simulated the edematous septic laryngitis seen in adults. On

admission, this case presented the question of a foreign body, also the possibility of an allergic reaction.

C. B., age 2½ years, entered the Maine General Hospital Nov. 28, and died on Nov. 28, 1935. The night before admission he had eaten grapes, refused his supper and was nauseated. Later that evening he had increasing laryngeal dyspnea. On entry to the hospital his temperature, pulse and respirations were 104, 160 and 40. On examination of the chest, an inspiratory stridor and expiratory flutter was noted. Chest X-rays were negative. A direct laryngoscopy was done at 9:00 A.M., and flaps of edematous tissue from the arytenoids overhung the glottis, causing the flutter heard on expiration. A tracheotomy was done over a 4 mm. bronchoscope. His breathing became easy, but he died of toxemia at 10:00 P.M. The lymph nodes about his neck became much swollen in course of the day. Autopsy revealed an inflamed and edematous larynx, marked subglottic edema with extension of inflammation and swelling down the tracheobronchial tree. There was no crusting or obstruction of the airways by edema. The lymph nodes of the neck were markedly swollen. Death was due to toxemia. Culture showed hemolytic streptococcus.

Case 13. Inadequate Larynx, Subsequent Laryngeal Dilatations. Recovery:

Comment: This was a case of acute laryngo-tracheobronchitis developing in a poorly nourished child with an inadequate larynx. The tracheotomy opening was through the second and third rings, and the cricoid cartilage was not injured. She eventually made a complete recovery.

J. G., age 18 months, entered the Maine General Hospital Nov. 18, 1937, and was discharged June 20, 1938. Since pertussis in July, she had had periods of cough, dyspnea and cyanosis. She was admitted to the hospital because of an upper respiratory infection and laryngeal dyspnea. Her temperature, pulse and respirations were 105R, 150 and 40. She was a poorly nourished baby of 20 pounds weight. Her larynx appeared normal at direct laryngoscopy in the morning, but that evening her dyspnea increased and a tracheotomy was performed. Her temperature gradually came down, but we were unable to leave the tracheotomy tube out until her larynx had been dilated seven times in a period of seven months. The original culture showed hemolytic streptococci and influenza bacilli.

Case 14. Delayed Tracheotomy. Death:

Comment: Tracheotomy was delayed at the suggestion of consultants; had it been done before the patient was exhausted, he would have had a better chance to recover.

W. F., age 13 years, entered St. Barnabas Hospital on Jan. 20, and died on Jan. 21, 1929. Four days before entry he had a mild sore throat, the next day a tonsillitis on the left, and the following day on the right. His tonsils were extremely large. He was sent into the hospital as his family were living at a hotel, not because he was considered to be particularly ill. On admission his temperature, pulse and respirations were 103.6, 114 and 24. That evening he began to have increasing dyspnea, although he had a clear voice. His tonsils were swollen but not abscessed, and met in midline and apparently caused his dyspnea. A tracheotomy was performed, after which he breathed more easily, but

died apparently of exhaustion the following day. He was an over-stout child, whose build suggested an endocrinopathy. He was given 20,000 units of diphtheria antitoxin. Culture showed staphylococcus aureus.

SUMMARY.

1. An acute laryngotracheobronchitis is a relatively uncommon disease, appearing most frequently in infants and children.

2. It tends to spread by continuity of tissue from the larynx and trachea to the smaller airways.

3. The secretions are viscid and as the disease progresses there is a tendency to the occlusion of the smaller airways by secretion, crusts and edema.

4. The hemolytic streptococcus is the organism most frequently found, but any of the common bacteria except the diphtheria bacillus may be recovered in pure or mixed culture.

5. Treatment demands bed rest, highly humidified air, adequate fluid intake and, at times, sulfanilamide, tracheotomy or intubation, instillation into the tracheotomy tube to prevent drying of secretions of normal saline, or, if the staphylococcus is present, bacteriophage, and bronchoscopic removal of secretion and crusts.

6. There are here reported the case histories of 14 patients on whom tracheotomy was performed because of the severity of the infection. The mortality was 50 per cent.

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47 Deering Street.

NEW YORK ACADEMY OF MEDICINE.

Regular Meeting of May 15, 1940.

SECTION ON OTOLARYNGOLOGY.

Otogenous Tetanus. Dr. Samuel Rosen.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Thrombophlebitis of the Cavernous Sinus of Otitic Origin, with Report of Case. Dr. Joseph G. Druss.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

The "Blocked Ear" of the Caisson Worker. Dr. Ralph Almour.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. JOHN D. KERNAN: It is certainly a very important subject, although most of us do not see caisson workers. With the advance of air travel it is certain that more and more people will be affected in some degree when they fly. I was going to ask about that, Dr. Almour, if you hadn't mentioned it yourself, and about aviators. We are going to see a great many of those cases and I think they are probably more important than the caisson case itself.

DR. CHARLES J. IMPERATORI: I would like to ask Dr. Almour if he has any data in relation to sailors in submarine divisions.

DR. KERNAN: Do those cases that have hemorrhage recover more slowly than those in the first degree classification?

DR. RALPH ALMOUR: In regard to Dr. Imperatori's question, I think that the statistics on divers and submarine workers are available as far as the navy is concerned, because they can and do keep a check on their men. They have a record of their hearing before they go into this work and comparisons can be made, at least with what was normal for a particular man. But as far as the men are concerned who are working on our tunnels under the rivers, and on our subways, it is almost impossible to get a record, because, as I say, the union officials think we are trying to discriminate in employing the men and that we might be using the tests as a pretense for not hiring them.

As for Dr. Kernan's question, I would say, no — the intratympanic hemorrhage may clear up just as quickly as a hemorrhage not into the drum itself; and sometimes loss of hearing from the mildest form may be more persistent than hearing loss following a quite severe intratympanic hemorrhage. There is no characteristic that you can put your hand on, as far as testing is concerned, that would differentiate for you an intratympanic hemorrhage from one of mere injection of the drum, or one small hemorrhage from multiple hemorrhages into the membrane itself.

DR. KERNAN: Are cases that have a blocked ear more likely to have an intracochlear hemorrhage, too?

DR. ALMOUR: No: intracochlear hemorrhages occur in real caisson disease; that is, "the bends," and we get them only on decompression. The otologist doesn't see those cases except in the last stages, after the man has been put back into his lock, recompressed, slowly decompressed. When labyrinthine symptoms will appear, they incapacitate him, and the otologist gets him weeks or months after the injury. The report sent with the man may

state merely "hemorrhage from the ear" without any examination having been made.

Question: I want to ask whether in any of the 74 cases Dr. Almour saw, there were labyrinthine symptoms as a complaint?

DR. ALMOUR: Not in these cases.

DR. IMPERATORI: Any tinnitus?

DR. ALMOUR: At times — very little.

Atypical Facial Neuralgia. Dr. George H. Hyslop.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. RICHARD M. BRICKNER: Dr. Hyslop's paper is of special interest to me for a number of reasons. Although there are many phases of it which can be discussed, there is time only for a few. These are, first, atypical trigeminal neuralgia, which is the title of his paper and which name covers a rather widespread group of cases — really those in which the syndrome does not fit into the various other syndromes which include pain in the head and neck. One can start the examination of this question with classical, true migraine, about which there can be no dispute. In migraine there is pain always in the head itself and not in the face, and the sympathetic nervous symptoms are those of the general and not focal. They include nausea, vomiting, sometimes sweating, generalized pallor of the face, and so forth.

Then there is the group which originally received the name atypical trigeminal neuralgia. The manner of its discovery is of some interest. In the early days, Frazier, at the suggestion of Spilles, found that pain in the face might be cured by section of the trigeminal nerve. After a good many cases had been so treated, a small number were left over who were not helped. Since they were seen in a neuralgia clinic, they came to be known as cases of atypical neuralgia; however, they generally resembled trigeminal neuralgia a little more than the cases Dr. Hyslop has described, because in the cases he had, the pain was not limited to the trigeminal domain.

Vallery-Radot, the son-in-law of Pasteur, who wrote Pasteur's biography, was interested in migraine, seeing his patients in what must be called a migraine clinic. When he encountered cases similar to those of Frazier, he called them atypical migraine, not neuralgia. We, in our own migraine clinic, met such cases now and then, and in the beginning we, too, called them atypical migraine. It occurred to Dr. Riley and myself that it would be better to stop classifying them as atypical of something else, and to think of them as existing in their own right — to take away the bar sinister, as it were. Hence, we assigned to them the descriptive appellation "autonomic faciocephalalgia." We included instances of pain in the head and face, with focal sympathetic disturbances on the painful side of the face; moreover, we found that the symptoms could be relieved either by adrenalin or ergotamine.

Summarizing the kinds of cases with focalized autonomic disturbance, we have, then, first, autonomic faciocephalalgia, in which the sympathetic phenomena have become focalized. The next group, to which Dr. Hyslop alluded, is that with focalized sympathetic nervous disturbances in other parts of the body. The last group is one which we all see and which is always a curiosity. I refer to the painless cases of autonomic disturbance in the face, such as sweating in response to certain stimuli. It may occur when the patient eats something sour or when the face is suddenly cooled.

I will be very brief and say only a few more words. What we do know about the causes of these states is, unfortunately, little. Two of the most commonly invoked precipitators may be mentioned — allergy and psychoneurosis. I have never seen a case of this particular kind due to allergy. Some

of the migraines and other pains in the head itself may be, but not cases of this variety insofar as we know.

Of particular interest to all of us are cases which clearly include a psychological element. The problem always arises as to whether the whole picture is not psychogenic. One good rule of differential diagnosis is, if it is psychoneurotic the symptoms are likely to be in a place conceived of as an organ, an entity, such as the head, or an arm or leg, which the patient knows about and can use for the purpose of localizing his symptoms; but when the symptoms are localized in an anatomical area which as such is apt to be of little or no significance to the patient, the chances are against their being actually of psychological origin.

Of interest to us are those cases with disturbances in tissue, which act as an engraved invitation to the mind that that is the place to implant psychological symptoms. We may see this nicely illustrated in cases of Raynaud's disease. The spasms that produce the blanching of the fingers can be produced by cold, but I know of two cases where it could be produced by the thought of cold. In one, the fingers became blanched when the hand was placed over a basin of ice.

I would like to mention also an example of the other variety of situation, where, without an inviting lesion, a patient localizes a psychological disturbance in an organ. One was so unusual that I think it is worthy of note. The patient was an admitted psychoneurotic, who complained of itching in the groin for which no cause could be found. While on the examining table, the patient suddenly exclaimed that he felt the itching. It was found that the dartos was in violent contraction. The consequent movement of the hairs was directly responsible for the itching. Subsequently it was discovered that the itching, as well as the movement of the dartos, occurred every time the patient spoke of his mother.

The treatment of the cases Dr. Hyslop has described is very limited, as Dr. Hyslop said. The autonomic faciocephalalgia cases may indeed respond well to adrenalin or ergotamine tartrate. Sphenopalatine cocainization may also be employed. It might be worth while to mention that in our migraine clinic some 60 patients with true migraine were given cocainization without relief. Penfield, Craig, Glaser and others advocate widespread and radical sympathetic surgery, reporting considerable success.

Perhaps the most important point in Dr. Hyslop's paper centered around the man who had carcinoma of the tonsil with metastases to the glands of the neck. This physical lesion reproduced the syndrome of atypical neuralgia, or of autonomic faciocephalalgia. More cases of that type will really teach us how the syndrome is produced, and where and what the pathways are.

Dr. George H. Hyslop: One thing hasn't been mentioned — some of these people, by the time they get around to the neurologist's doorstep, have become pests. Everybody regards them as neurotics of some variety. I quoted Lewy and Grant in distinguishing people with the type of pain I described from those with true tic douloureux. True tic douloureux patients are, by and large, individuals who forget pain when it is not there. These atypical neuralgia patients are so made that they have it on their minds all the time, and you can't blame the doctor for thinking there is nothing wrong, or that it doesn't amount to anything. People with tic douloureux were once written about as people whose pain was some form of mental indigestion and it wasn't until we got busy and learned how to do surgery on the trigeminal roots that the psychiatrists stopped talking about such patients as being neurotics. Atypical facial neuralgia sufferers at the present stage are often regarded as neurotic because physicians still do not know what to do to help them.

I am appreciative of Dr. Bricker's competent discussion. In particular, his reference to what one might call the spectrum of face pain is useful. Differentiation between the several allied and, at times, overlapping of face and neck pain is not easy.

Injuries of the Larynx, Traumatic and Therapeutic. Dr. John D. Kernan.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. CHARLES J. IMPERATORI: It is very important in these patients having trauma of the larynx to recognize that they have a perichondritis to a greater or lesser degree. The resulting deformity alters the interior of the larynx. The subglottic area, spoken of by Dr. Kernan as the conus elasticus, is particularly vulnerable. Because of the structure, this area easily becomes infiltrated following an injury, with a resulting acute stenosis. In these acute conditions with definite signs of obstruction, to wait too long in doing a tracheotomy may prove disastrous. Complete obstruction may occur very suddenly.

A case history relating to laryngeal fracture was that of a young man, a gangster, who was being interrogated by the police — not the police of New York. He was thrown to the floor and one of the officers placed his instep on the patient's neck and stepped on him, figuratively and literally. Then there were administered a series of hose treatments, both with water in the hose and without, so that the man became unconscious. He was then thrown in the shower bath and left there, and, to their amazement, when they went back in about 15 minutes the man was dead. Autopsy showed fracture of the cricoid and one ala of the thyroid, with tremendous edema. Now, if the physician who was called had properly examined him and done a tracheotomy, it might have saved his life.

In an attempt to relieve an acute laryngeal stenosis resulting from a fracture of the cartilages, one should do a tracheotomy, then a thyrotomy, elevating the fractured cartilages into place, and splint the interior of the larynx with not too large an intubation tube. Great care must be exercised when using an intubation tube that it be not too large, otherwise pressure necrosis will result.

The dictum of Chevalier Jackson in these types of injuries of the larynx is "early tracheotomy and low," just as Dr. Kernan said.

My experience with core molds has been very satisfactory. Two years ago I saw a young man, age 18 years, with complete stenosis of the larynx. As a child of 4 years he had papilloma of the larynx and was treated by the application of radium to the larynx, both externally and internally. Eventually the stenosis became so marked that a tracheotomy was done. A famous laryngologist, whose country is now dominated by Hitler, while on a visit here in the United States did his operation. Unfortunately, it was a complete failure. The operation was completed by an associate during my absence on a vacation and this, too, resulted in a failure. The lumen of the larynx was completely obliterated. The procedure in this young man was to bore a hole through the scar tissue and pull a small core mold into the larynx. Increasing sizes of core molds were used so that he can now breathe through his larynx with the tracheotomy opening closed. In the use of core molds, one should not use too large sizes. The desideratum is dilatation with epithelization.

My experience with X-ray therapy in carcinoma of the larynx and the usually resulting perichondritis leads me to disagree with that form of treatment. I disagree because my own results have been very poor in radiation therapy of cancer of the larynx. We have a very good method of treatment in laryngofissure for a unilateral growth of the larynx; however, I feel that radiation will eventually be our best therapeutic measure.

I just want to say a few words about laminographs. My experience with them to date has been slightly unsatisfactory, the unsatisfactory part being that in several instances the growth was not visualized in the laminograph. Biopsy definitely proved we had a malignant growth. When a laminograph gives a positive report we should consider it, but if it does not, and clinical

signs indicate a growth, it should be considered as a negative finding. Where there is very little airway in the larynx, the laminograph helps but little.

DR. JAMES W. BABCOCK: I am very sorry Dr. Kernan did not include one case, that of a boy who had no larynx at all. I don't remember how he acquired that defect.

DR. KERNAN: Diphtheria, with tracheotomy through his larynx.

DR. BABCOCK: There was not a single cartilage left. Dr. Kernan made him a new larynx.

DR. KERNAN: I will say that Dr. Imperatori can't get a quarrel with me on the laminograph. I think it is a very interesting machine—we see a lot of shadows and we don't know what causes them. I do think it shows up a narrow lumen nicely.

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Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Broncho-Esophagological Association.

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.

American Laryngological Association.

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Los Angeles. Time: June 16-18, 1941.

Sections:

Eastern—Chairman: Dr. N. S. Weinberger, Robert Packer Hospital, Sayre, Pa.
Southern—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville.
Middle—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.
Western—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology.

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland. Time: June 2-6, 1941.

Pacific Coast Oto-Ophthalmological Society.

President: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Los Angeles. Time: May 26-29, 1941.